

# **Neonatal Brachial Plexus Palsy**



**The American College of  
Obstetricians and Gynecologists**  
WOMEN'S HEALTH CARE PHYSICIANS

*Neonatal Brachial Plexus Palsy* was developed under the direction of the American College of Obstetricians and Gynecologists' Task Force on Neonatal Brachial Plexus Palsy. The information in *Neonatal Brachial Plexus Palsy* should not be viewed as a body of rigid rules. The information is general and intended to be adapted to many different situations, taking into account the needs and resources particular to the locality, the institution, or the type of practice. Variations and innovations that improve the quality of patient care are to be encouraged rather than restricted. The objectives of this report will be met if it provides a firm basis on which local norms may be established.

Studies were reviewed and evaluated for quality according to the method outlined by the U.S. Preventive Services Task Force (USPSTF):

- I Evidence obtained from at least one properly designed randomized controlled trial
- II-1 Evidence obtained from well-designed controlled trials without randomization
- II-2 Evidence obtained from well-designed cohort or case-control analytic studies, preferably from more than one center or research group
- II-3 Evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled experiments could also be regarded as this type of evidence.
- III Opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees

Although there is no independent grading system of the biomedical engineering literature analogous to that of the USPSTF quality guidelines for clinical studies, articles published in this field are subject to standard peer-review processes.

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# Foreword

**N**eonatal brachial plexus palsy (NBPP) is a rare occurrence, with an overall incidence of 1.5 per 1,000 total births. Favorable outcomes or complete recovery are variously estimated as low as 50% and as high as 80%, considering all types of lesions. Although NBPP is sometimes associated with shoulder dystocia, it is well recognized that NBPP also occurs without concomitant, clinically recognizable shoulder dystocia.

I thought it would be most useful, not only to our Fellows, but to all those who provide maternal and neonatal care, to provide an updated resource on this subject. I had in mind a report, based on a critical appraisal of the relevant peer-reviewed literature, on the existing state of knowledge of the etiology, as well as the prediction, management, and treatment of NBPP.

Robert Gherman, MD, and the members of the Task Force on Neonatal Brachial Plexus Palsy have written this report that addresses my objectives. Recent multidisciplinary research now stresses that the existence of NBPP following birth does not *a priori* indicate that exogenous forces are the sole cause of this injury. And, in the presence of shoulder dystocia, all intervention by way of ancillary maneuvers—no matter how expertly performed—will necessarily increase strain on the brachial plexus.

In closing, I would like to thank the task force and Albert Strunk, JD, MD, deputy executive vice president of the American College of Obstetricians and Gynecologists, for providing a well-researched, informative, objective, and dispassionate presentation of the existing state of knowledge on this subject. I hope you will find this information to be of help in both patient education and clinical management.

James T. Breeden, MD  
President  
The American College of Obstetricians and Gynecologists, 2012–2013  
The American Congress of Obstetricians and Gynecologists, 2012–2013



# Preface

In 2011, James T. Breeden, MD, then president-elect of the American College of Obstetricians and Gynecologists (the College) called for the formation of a task force on neonatal brachial plexus palsy (NBPP). A working group on NBPP convened in November 2011. It comprised nine subject-matter experts: six maternal-fetal medicine specialists, a neonatologist, a neurosurgeon, a biomechanical engineer, and a liaison member from the Society of Obstetricians and Gynaecologists of Canada. Four of the maternal-fetal medicine specialists, Robert B. Gherman, MD; Bernard Gonik, MD; Suneet P. Chauhan, MD; and Joseph G. Ouzounian, MD; have contributed extensively to the peer-reviewed literature on NBPP and shoulder dystocia. The fifth maternal-fetal medicine specialist, Steven L. Clark, MD, was chosen for his expertise and research in critical care obstetrics, fetal evaluation, and patient safety. The sixth maternal-fetal medicine specialist, William A. Grobman, MD, conducts research and writes on patient safety and the provision of high-quality obstetric care. The clinical practice and research of the neurosurgeon, Lynda J.-S. Yang, MD, focuses on the treatment and outcomes of pediatric and adult patients with brachial plexus and peripheral nerve injuries. The research of the biomechanical engineer, Michele J. Grimm, PhD, centers on tissue biomechanics, ranging from brain to bone, with a particular interest in injury biomechanics. The neonatologist, Jay P. Goldsmith, MD, provided expertise on neonatal evaluation, hypoxia-ischemia, resuscitation, and ventilation, as well as performance improvement.

On Dr. Breeden's accession to College President, the working group was formally constituted as a task force, with the following charge:

To review and summarize the current state of the scientific knowledge, as set forth in the peer-reviewed and relevant historical literature, about the mechanisms which may result in neonatal brachial plexus palsy. The purpose of conducting such review is to produce a report which will succinctly summarize the relevant research on the pathophysiology of neonatal brachial plexus palsy. Although primarily intended to inform the College's Fellows about the existing state of knowledge as to the etiology, as well the prediction, management, and treatment of neonatal brachial plexus palsy, this report also is meant to serve as a resource for all health care providers involved in this subject matter area.

At the first meeting, members outlined the subject matter to be covered; agreed on assigned topics; and established a program for review, critical appraisal, and verification of materials, methods, and conclusions. A subgroup of the task force met again in November 2012 and February 2013. Multiple conference calls were held as needed to advance the work of the task force. Independent reviewers were asked to consider the final draft and contribute their opinions and expertise.

## Methods

The task force members, as well as College staff, retrieved published literature after searches of MEDLINE, the Cochrane Library, and other databases (such as OVID), as well as the College's own internal resources. Searches were updated on a regular basis and incorporated in this report. The search was primarily restricted to articles published in English, although non-English literature of historic significance was included. Priority was given to articles reporting results of original research, although review articles and commentaries also were consulted. In addition, targeted searches in the biomedical literature revealed significant relevant material, some of which involves the use of physical and computer modeling, as well as cadaveric and animal studies. Guidelines published by organizations or institutions such as the National Institutes of Health were reviewed, and additional studies were located by reviewing the bibliographies of identified articles. Abstracts of research presented at symposia and scientific conferences were not considered adequate for inclusion.

Studies were reviewed and evaluated for quality according to the method outlined by the U.S. Preventive Services Task Force (USPSTF):

- I Evidence obtained from at least one properly designed randomized controlled trial
- II-1 Evidence obtained from well-designed controlled trials without randomization
- II-2 Evidence obtained from well-designed cohort or case-control analytic studies, preferably from more than one center or research group
- II-3 Evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled experiments could also be regarded as this type of evidence.
- III Opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees

Although there is no independent grading system of the biomedical engineering literature analogous to that of the USPSTF quality guidelines for clinical studies, articles published in this field are subject to standard peer-review processes.

## Task Force Objectives

The task force was directed to answer, if possible, the following questions:

- What is the incidence of NBPP?
- Can NBPP be reliably predicted and prevented?
- What is the pathophysiologic mechanism for NBPP with and without shoulder dystocia?
- What are the endogenous and exogenous forces inherent in the process of labor and delivery that may cause or contribute to the development of NBPP?
- Does simulation aid in training health care providers and improve outcomes when clinical shoulder dystocia occurs?
- What mechanisms for the etiology of NBPP are offered by the peer-reviewed literature?

- What are the biomechanic effects of maneuvers commonly used to alleviate recognized shoulder dystocia?
- How does NBPP clinically present?
- Are there early treatments or later surgical or nonoperative interventions that can ameliorate the disability of NBPP?
- What are the expected recovery rates for NBPP?
- Are there specific interventions that may reduce the frequency of NBPP?

The document is organized around these questions.

## **Association of Neonatal Brachial Plexus Palsy With Shoulder Dystocia**

Shoulder dystocia, in the context of clinical obstetrics, is said to result from a lack of fit or accommodation of the transverse diameter of the fetal shoulders to the several pelvic diameters traversed by the fetus in the course of labor and delivery. This disproportion can be the result of excessive fetal size or inadequate pelvic capacity. Although abnormalities of presentation, position, or development of the fetus, as well as abnormalities of the maternal bony pelvis or the soft tissues of the reproductive tract or both, may result in shoulder dystocia, most reported cases do not involve these factors.

Inasmuch as a transient or protracted arrest of descent of the fetal shoulder can involve the anterior or posterior or both shoulders, it also is necessary to distinguish between shoulder dystocia resulting from arrest of the fetus' anterior shoulder behind the symphysis pubis and dystocia resulting from arrest of the posterior shoulder at the level of the sacral promontory or in the hollow of the pelvis. The former is clinically evident on delivery of the fetus' head, whereas the latter occurs before delivery of the fetus' head and is not clinically apparent or ascertainable at the time of its occurrence. In both instances it is the arrest of descent of the affected shoulder, despite the ongoing downward movement of the axial skeleton, which widens the angle between neck and impacted shoulder and stretches the brachial plexus.

This report represents the culmination of the task force's effort and was written in 2013 in collaboration with the Society of Obstetricians and Gynaecologists of Canada. The task force recognizes that the subject matter of this report is an area of continually evolving research and knowledge. As new evidence becomes available, scientific information and clinical guidance will be updated through College Practice Bulletins and Committee Opinions to ensure that the work of the task force remains current.

This report reflects the engagement and commitment of the task force members who gave unstintingly of their time and expertise in analyzing the literature and writing the document's six chapters, as well as in reviewing the work of other members and outside reviewers. Vyta Senikas, MD, MBA, FRCSC, liaison member representing the Society of Obstetricians and Gynaecologists of Canada, provided insightful critique and expert editorial guidance. Independent reviewers Ronald T. Burkman, MD; Nancy C. Chescheir, MD; Washington C. Hill, MD; Joseph E. Hornyak, MD, PhD; Amy J. Houtrow, MD, PhD, MPH; Linda J. Michaud, MD; Virginia S. Nelson, MD, MPH; Dwight J. Rouse, MD; James R. Scott, MD; and Earl T. Stubblefield, MD, gave an impartial perspective and commentary on both

form and substance of the report. Jeffrey Klagholz of the College bore responsibility for reconciling the various drafts from task force members, maintaining the document's updated master file, and managing the task force's workflow. James Lumalcuri, American College of Obstetricians and Gynecologists' senior director, provided managerial oversight and support. Dana Trevas, independent developmental editor, offered new perspective and numerous edits that greatly improved the report's organization and clarity. Albert Strunk, JD, MD, American College of Obstetricians and Gynecologists' deputy executive vice president, served as the staff physician liaison to the task force and contributed extensively and in detail to the editing and revision of virtually every section of the document. The combined effort of the task force members, liaison members, independent reviewers, independent developmental editor, and College staff is gratefully acknowledged.

## **Endorsements**

The March of Dimes Foundation and The Royal College of Obstetricians and Gynaecologists have offered their full support of this report. In addition, the following professional organizations have endorsed this report:

- The American Academy of Pediatrics
- The American Academy of Physical Medicine and Rehabilitation
- The American College of Nurse-Midwives
- The American Gynecological and Obstetrical Society
- The American Society for Reproductive Medicine
- The Child Neurology Society
- The Japan Society of Obstetrics and Gynecology
- The Royal Australian and New Zealand College of Obstetricians and Gynaecologists
- The Society for Maternal-Fetal Medicine
- The Society of Obstetricians and Gynaecologists of Canada

All task force members provided disclosure statements. No material, financial, business, or other interest that would compromise an author's ability to undertake unbiased scientific research and writing was declared.

Robert B. Gherman, MD  
Chair, Task Force on Neonatal Brachial Plexus Palsy  
July 2013

# **Executive Summary**

The American College of Obstetricians and Gynecologists convened the Task Force on Neonatal Brachial Plexus Palsy to develop a comprehensive report summarizing the scientific literature on this subject. Clinically, neonatal brachial plexus palsy (NBPP) presents in a newborn as a weak or paralyzed upper extremity, with the passive range of motion greater than the active. The overall incidence of NBPP, both transient and persistent impairment, is 1.5 per 1,000 total births. Multiple reports in the peer-reviewed literature describe the occurrence of NBPP without concomitant clinically recognizable shoulder dystocia at the time of both vaginal and cesarean delivery. Chapter 1 of the report details the incidence of NBPP.

## **Risk Factors**

Various risk factors have been described in association with NBPP. They include fetal mal-position, labor induction, labor abnormalities, operative vaginal delivery, fetal macrosomia, and shoulder dystocia. Overall, except for shoulder dystocia, these risk factors have not been shown to be statistically significant or clinically useful predictors for the occurrence of NBPP. Most NBPP cases (greater than 80%) occur in women without known risk factors. Risk factors for shoulder dystocia are not reliable predictors for its occurrence or the occurrence of NBPP. Thus, no intervention has been identified that will prevent all or even most cases of NBPP or clinically apparent shoulder dystocia.

Notwithstanding the unreliability of specific risk factors to predict NBPP or clinically apparent shoulder dystocia in a specific case, there are three clinical situations of concern to practitioners in which an alteration of usual obstetric management might be considered to reduce the risk of shoulder dystocia and brachial plexus injury: 1) suspected fetal macrosomia with estimated fetal weight exceeding 5,000 g in women without diabetes or 4,500 g in women with diabetes; 2) prior recognized shoulder dystocia, especially with a severe neonatal injury; and 3) midpelvic operative vaginal delivery with fetal birth weight more than 4,000 g. Even in these circumstances, the occurrence of NBPP is relatively low, and with proper informed consent, numerous clinical situations exist in which these risk factors alone should not dictate a particular course of management. Chapter 2 discusses risk factors in more depth.

## Causation

Chapter 3 delves into the pathophysiology and biomechanics of NBPP. Uterine contractions and maternal pushing efforts produce pressure within the uterus that is transmitted by direct contact and through amniotic fluid. This pressure produces a force that moves the fetus into and through the pelvis. Both maternal (endogenous) forces and clinician-applied (exogenous) forces have a direct effect on the fetus as a whole and on its discrete anatomic structures. Maternal forces alone are an accepted cause of at least transient NBPP by most investigators. In addition, NBPP can be caused by downward lateral traction applied by the birth attendant. Downward lateral traction (bending of the fetus' neck away from the anterior shoulder, toward the posterior shoulder or floor) has been shown to place greater strain on the brachial plexus than downward axial traction, wherein the applied forces are parallel to the fetus' cervicothoracic spine. Nonetheless, even properly applied axial traction can result in NBPP, as discussed further in Chapter 3.

Neither high-quality nor consistent data exist to suggest that NBPP can be caused only by a specific amount of applied force beyond that typically used by health care providers during any delivery. Instead, available data suggest that the occurrence of NBPP is a complex event, dependent not only on the forces applied at the moment of delivery, but also on the constellation of forces (eg, vector and rate of application) that have been acting on the fetus during the labor and delivery process, as well as individual fetal tissue characteristics (eg, in situ strain and acid-base balance). In addition to research within the obstetric community, the pediatric, orthopedic, and neurologic literature now stress that the existence of NBPP following birth does not a priori indicate that exogenous forces are the cause of this injury.

## Shoulder Dystocia and Neonatal Brachial Plexus Palsy

Chapter 4 addresses the association of shoulder dystocia and NBPP. A commonly accepted definition of shoulder dystocia is a delivery that requires additional obstetric maneuvers following failure of gentle downward traction on the fetal head to effect delivery of the shoulders. Clinically, shoulder dystocia is diagnosed after delivery of the fetal head when the anterior shoulder fails to emerge under the pubic rami as a result of impaction of the fetus' anterior shoulder behind the pubic symphysis. Although in most cases, a combination of factors raises suspicion that shoulder impaction against the pubic symphysis has occurred, shoulder dystocia is not formally diagnosed until a trial of downward axial traction has been unsuccessful in delivering the anterior shoulder. Recognized shoulder dystocia occurs infrequently, with an incidence ranging from 0.2% to 3.0% of all vaginal deliveries. It appears that the incidence of clinically evident shoulder dystocia has been increasing over the past several decades. Nonetheless, the incidence of NBPP has remained relatively constant over time. Current and emerging approaches to the management of shoulder dystocia also are discussed in Chapter 4 and further detailed in the appendixes.

## Anatomy, Presentation, and Assessment

The brachial plexus is a very complex structure that connects the spinal nerves to their terminal branches in the upper extremity. Understanding the schematic anatomy of the nerves and the muscles they innervate provides a critical foundation from which to understand the clinical presentation of NBPP. Anatomic variation of the plexus can occur among the spinal roots that contribute to the brachial plexus and can subsequently

affect the clinical presentation. Chapter 5 describes factors affecting the clinical presentation of NBPP and gives a detailed summary of anatomic considerations.

Determining when an injury to the neonatal brachial plexus occurred may have prognostic as well as medicolegal implications; therefore, the timing should be assessed whenever possible and relevant events and findings thoroughly documented. Birth attendants may suspect NBPP on the basis of initial observations of the neonate. However, the ultimate diagnosis is best achieved by the combined efforts of neonatologists, neurologists, pediatricians, physiatrists, and occupational and physical therapists. If NBPP is suspected, the treating clinician should assess the infant for skeletal injuries and fractures by clinical and radiographic examination because some musculoskeletal injuries preclude early therapy for NBPP. Components of the physical examination and assessments are described in Chapter 5. Assessment scales of NBPP are used to gauge the extent of injury, prognosticate potential recovery, and determine further treatment, as well as evaluate overall function.

## Patterns and Early Intervention

Chapter 6 describes patterns of NBPP. In NBPP, the upper plexus is affected more frequently (Erb palsy) than either the lower plexus alone (classic Klumpke palsy) or the complete nerve complex. The natural history of NBPP, around which the determination of optimal treatment revolves, remains the subject of debate in many published reports. The complete potential scope of NBPP is difficult to define because of the various combinations of lesions within the elements of the brachial plexus. Further difficulties include the definition of recovery and the potential bias introduced by the referral patterns of reporting physicians because many patients with Erb palsy recover spon-

taneously and are not referred to the specialists who publish most reports. Whereas early clinical improvement is ultimately associated with favorable outcomes, most practitioners agree that as an absence of spontaneous clinical improvement persists, the potential for recovery diminishes.

Chapter 6 goes on to address indications for surgery for NBPP. The indications for surgical nerve reconstruction in NBPP vary widely among different practitioners. However, most agree that early nerve reconstruction should be recommended for panplexopathy and preganglionic nerve root lesions. Overall, infants who sustain NBPP have a good prognosis, with the majority recovering adequate functional use of the affected arm without surgical intervention. Early occupational and physical therapy can support spontaneous recovery of function and minimize musculoskeletal comorbidities. Given recent and continuing improvements in surgical and conservative treatments, early referral to a specialty care center can improve overall outcomes after NBPP.

## Summary

The report reviews the peer-reviewed and other pertinent literature on NBPP with special emphasis on its pathophysiology and causation. Some strategies that demonstrate either a reduction in NBPP or an increased rate of successful resolution of shoulder dystocia are included, but the work does not present comprehensive management strategies. Moreover, no single management strategy is viewed as preferable.

The primary objective in the presence of clinically recognizable shoulder dystocia continues to be the delivery of the fetus before the fetal brain experiences hypoxic-ischemic injury. Any intervention to effect delivery must necessarily balance the risk of using ancillary maneuvers, which will increase strain on the fetus' brachial plexus, against the risk of hypoxic-ischemic brain injury.

The birth attendant is the individual best equipped to assess this balance and decide on the type and degree of intervention.

Significant advances have been made in the science of biomedical engineering that have informed this document and will continue to increase the understanding of the mechanical forces that affect the fetus during labor and delivery. In the years to come, the greater sophistication of physical and computer models will enhance the ability to assess the effects of both endogenous forces of labor and delivery and exogenous forces generated by the birth attendant in facilitating an obstructed delivery.

The task force recognizes that knowledge about NBPP is continually evolving. What is known at this time with reasonable medical certainty is that NBPP occurs infrequently and can be caused by maternal (endogenous) forces or clinician-applied (exogenous) forces or a combination of both. Similarly, NBPP can occur with or without associated, clinically recognizable shoulder dystocia. Finally, in the presence of shoulder dystocia, all intervention by way of ancillary maneuvers—no matter how expertly performed—will necessarily increase strain on the brachial plexus.

# **Neonatal Brachial Plexus Palsy**



# Chapter 1

## Incidence of Neonatal Brachial Plexus Palsy

**N**eonatal brachial plexus palsy (NBPP) presents in a newborn as a weak or paralyzed upper extremity, with the passive range of motion greater than the active. Brachial plexus injuries are classically defined as Erb palsy or Klumpke palsy. Erb (upper) palsy involves C-5 and C-6 cervical nerve roots. It is recognizable by the characteristic “waiter’s tip” arm position caused by muscle imbalance that holds the shoulder in an adducted, internally rotated position with the elbow in extension and forearm in pronation. Klumpke palsy reflects damage to the lower cervical and upper thoracic nerve roots (C-8 and T-1). Findings typically associated with Klumpke palsy are weakness of the hand and medial forearm muscles. Chapter 5 provides a more complete description of anatomic factors.

Most studies in the obstetric literature use a clinical definition of NBPP to include some degree of neurologic dysfunction to the neonate’s arm. Interpreting the literature is complicated by the following: 1) lack of confirmation of clinically suspicious brachial plexus injuries by pediatric neurologic examination or imaging; 2) limited or absent description of long-term follow-up; 3) lack of review of medical, surgical, and occupational and physical therapy records to determine whether

or when the neurologic disability resolved; and 4) lack of a standard definition of what constitutes a persistent injury. Brachial plexus injuries associated with a component of residual neurologic dysfunction are defined as persistent. Persistent brachial plexus injury, as described in the obstetric literature, refers to those injuries associated with a component of residual neurologic dysfunction 12 or more months after birth.

Recognizing the limitations of a retrospective review of population-based studies, one can nevertheless approximate the frequency of NBPP. On the basis of cumulative data from 40 publications of retrospective reviews (Table 1-1), the incidence of NBPP, both transient and persistent, is 1.5 per 1,000 total births (1–40). The overall incidence of NBPP in U.S. studies is 1.5 per 1,000 total births (range: 0.7–8.1/1,000 total births), compared with 1.3 per 1,000 total births (range: 0.1–3.6/1,000 total births) in other countries (Table 1-1). Among the 40 publications cited, 15 provide data on vaginal births and NBPP. In these 15 reports, the overall prevalence was 1.7 per 1,000 vaginal births (4–6, 8, 11, 13, 15, 19, 20, 23, 24, 32, 35, 37, 38). In absolute terms, rates in the United States have varied little in the past 20 years, from 1.2 per 1,000 births in studies conducted before 1990 to 1.5 per

1,000 births in studies conducted since 1999. In these studies, NBPP included both transient and persistent conditions. Fifteen reports address the issue of transient versus persistent injury. All 15 reports monitored the affected neonates for a minimum of 12 months. Rates of persistent NBPP at 12 months following birth ranged from 3% to 33% ([Table 1-2](#)). As evidenced in [Table 1-3](#), Erb palsy represents the majority of cases of shoulder dystocia-related brachial plexus palsy (1.2/1,000 total births), while Klumpke palsy occurs exceedingly infrequently (0.05/1,000 total births).

### **Incidence of Neonatal Brachial Plexus Palsy Without Shoulder Dystocia**

Twelve reports published between 1990 and 2011 have described NBPP without concomitant shoulder dystocia (3, 4, 12, 13, 21–23, 29, 32, 33, 36, 41). One of the earliest reports (1990) was from Finland, where the authors noted 16 cases of NBPP among 14,000 total births, one half of which had no documented shoulder dystocia (21). A summary of these 12 reports from six countries (Finland, Ireland, Norway, Sweden, the United States, and the United Kingdom) indicates that 46% (1,445/3,111) of NBPP occurred without documented concomitant shoulder dystocia ([Table 1-4](#)). From these reports, the rate of NBPP without shoulder dystocia can be estimated as approximately 0.9 per 1,000 births. It is noteworthy that two of these reports provide data

on persistent NBPP without shoulder dystocia (13, 42). Even when the injury to the brachial plexus was documented as lasting more than 1 year, 26% (49/191) occurred in the absence of documented shoulder dystocia. Few investigators have attempted to describe whether the frequency of Erb palsy or Klumpke palsy varies depending on the presence or absence of shoulder dystocia.

### **Incidence of Neonatal Brachial Plexus Palsy Associated With Cesarean Delivery**

Six publications include data to support the calculation of the number of cases of NBPP with cesarean delivery ([Table 1-5](#)) (6, 11, 13, 23, 30, 42). In the four U.S. studies that evaluated more than 240,000 births, the rates range from 0.3 per 1,000 live births to 1.5 per 1,000 live births (6, 11, 13, 41). In the largest study of 216,000 births, the rate of NBPP was 0.3 per 1,000 cesarean births (11). The likelihood of NBPP with cesarean delivery in Ireland was 0.4 per 1,000 cesarean births (5 cases of NBPP among 11,526 cesarean deliveries) (23). In Singapore, the rate was 0.8 per 1,000 cesarean deliveries (4 cases of NBPP among 5,023 cesarean deliveries) (30). Publications on the topic are limited by insufficient information about the labor and the difficulty, if any, encountered during the delivery of the fetus through the uterine incision, as well as the presence or absence of soft tissue dystocia.

**Table 1–1.** Rate of Neonatal Brachial Plexus Palsy ↵

Authors	Publication date	Country	Study period	Total births	Number of cases of NBPP	Rate of NBPP (per 1,000 total births)
Rubin A <sup>1</sup>	1964	United States	1954–1959	15,435	18	1.2
Levine MG et al <sup>2</sup>	1984	United States	1974–1977, 1979–1981	13,870	36	2.6
Jennett RJ et al <sup>3</sup>	1992	United States	1977–1990	57,597	39	0.7
Nocon JJ et al <sup>4</sup>	1993	United States	1986–1990	14,297	33	2.3
Perlow JH et al <sup>5</sup>	1996	United States	1985–1990	19,370	17	1.1
Graham EM et al <sup>6</sup>	1997	United States	1987–1991	14,358	15	1.3
Ecker JL et al <sup>7</sup>	1997	United States	1985–1993	77,616	89	1.1
Gherman RB et al <sup>8</sup>	1998	United States	1991–1995	58,565	48	1.0
Bryant DR et al <sup>9</sup>	1998	United States	1984–1995	63,761	80	1.3
Turrentine MA, Ramirez MM <sup>10</sup>	1999	United States	1996–1998	3,008	6	2.0
Gilbert WA et al <sup>11</sup>	1999	United States	1994–1995	1,094,298	1,611	1.8
Chauhan SP et al <sup>12</sup>	2005	United States	1980–2002	89,978	89	1.0
Gurewitsch ED et al <sup>13</sup>	2006	United States	1993–2004	20,478	128	8.1
Weizsaecker K et al <sup>14</sup>	2007	United States	2000–2004	11,001	45	4.1
Chauhan SP et al <sup>15</sup>	2007	United States	2000–2004	41,200	38	0.9
Ford AA et al <sup>16</sup>	2007	United States	1998–2002	299,130	608	2.0
Foad SL et al <sup>17</sup>	2008	United States	1997, 2000, 2003	11,555,823	17,334	1.5
Grobman WA et al <sup>18</sup>	2011	United States	2005–2006	14,812	12	0.8
Inglis SR et al <sup>19</sup>	2011	United States	2003–2009	18,677	33	2.8
Paris AE et al <sup>20</sup>	2011	United States	1998–2009	94,842	102	1.5

(continued)

#### 4 Incidence of Neonatal Brachial Plexus Palsy

**Table 1–1.** Rate of Neonatal Brachial Plexus Palsy (*continued*)

Authors	Publication date	Country	Study period	Total births	Number of cases of NBPP	Rate of NBPP (per 1,000 total births)
<b>Foreign Countries</b>						
Salonen IS et al <sup>21</sup>	1990	Finland	1981–1987	14,265	16	1.1
Donnelly V et al <sup>22</sup>	2002	Ireland	1994–1998	35,796	54	
Walsh JM et al <sup>23</sup>	2011	Ireland	1994–1998; 2004–2008	77,624	121	1.6
Gonen R et al <sup>24</sup>	1996	Israel	1994–1995	4,480	11	2.8
Gonen R et al <sup>25</sup>	2000	Israel	1995–1999	16,416	17	1.0
Kees S et al <sup>26</sup>	2001	Israel	1996–1999	24,000	13	0.5
Bahar AM <sup>27</sup>	1996	Kuwait	1989	13,756	30	2.2
Soni AL et al <sup>28</sup>	1985	Libya	1983	7,829	28	3.6
Backe B et al <sup>29</sup>	2008	Norway	1991–2000	30,574	91	3.0
Tan KL <sup>30</sup>	1973	Singapore	1969–1971	90,436	57	0.6
Sjöberg I et al <sup>31</sup>	1988	Sweden	1973–1982	25,736	48	1.9
Gudmundsson S et al <sup>32</sup>	2005	Sweden	1990–1996	16,743	51	3.3
Mollberg M et al <sup>33</sup>	2005	Sweden	1987–1997	1,213,987	2,399	2
Dawodu A et al <sup>34</sup>	1997	United Arab Emirates	1993–1995	9,231	27	2.9
Olugbile A, Mascarenhas L <sup>35</sup>	2000	United Kingdom	1991–1995	28,932	2	0.1
Tandon S, Tandon V <sup>36</sup>	2005	United Kingdom	1989–1995	25,855	27	1.0
MacKenzie IZ et al <sup>37</sup>	2007	United Kingdom	1991–2005	95,321	44	0.6
Draycott TJ et al <sup>38</sup>	2008	United Kingdom	1996–1999; 2001–2004	39,220	30	1.0
Melendez J et al <sup>39</sup>	2009	United Kingdom	2000–2006	21,376	13	0.6
Evans-Jones G et al <sup>40</sup>	2003	United Kingdom & Ireland	1998–1999	776,618	323	0.4
<b>Total</b>				16,146,311	23,783	1.5

Abbreviation: NBPP, neonatal brachial plexus palsy.

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- <sup>8</sup> Gherman RB, Ouzounian JG, Goodwin TM. Obstetric maneuvers for shoulder dystocia and associated fetal morbidity. *Am J Obstet Gynecol* 1998;178:1126–30.
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- <sup>10</sup> Turrentine MA, Ramirez MM. Adverse perinatal events and subsequent cesarean rate. *Obstet Gynecol* 1999;94:185–8.
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- <sup>13</sup> Gurewitsch ED, Johnson E, Hamzehzadeh S, Allen RH. Risk factors for brachial plexus injury with and without shoulder dystocia. *Am J Obstet Gynecol* 2006;194:486–92.
- <sup>14</sup> Weizsaecker K, Deaver JE, Cohen WR. Labour characteristics and neonatal Erb's palsy. *BJOG* 2007;114:1003–9.
- <sup>15</sup> Chauhan SP, Cole J, Laye MR, Choi K, Sanderson M, Moore RC, et al. Shoulder dystocia with and without brachial plexus injury: experience from three centers. *Am J Perinatol* 2007;24:365–71.
- <sup>16</sup> Ford AA, Bateman BT, Simpson LL, Ratan RB. Nationwide data confirms absence of 'July phenomenon' in obstetrics: it's safe to deliver in July. *J Perinatol* 2007;27:73–6.
- <sup>17</sup> Foad SL, Mehlman CT, Ying J. The epidemiology of neonatal brachial plexus palsy in the United States. *J Bone Joint Surg Am* 2008;90:1258–64.
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- <sup>19</sup> Inglis SR, Feier N, Chetiyaar JB, Naylor MH, Sumersille M, Cervellione KL, et al. Effects of shoulder dystocia training on the incidence of brachial plexus injury. *Am J Obstet Gynecol* 2011;204:e1–6.
- <sup>20</sup> Paris AE, Greenberg JA, Ecker JL, McElrath TF. Is an episiotomy necessary with a shoulder dystocia? *Am J Obstet Gynecol* 2011;205:217.e1–3.
- <sup>21</sup> Salonen IS, Uusitalo R. Birth injuries: incidence and predisposing factors. *Z Kinderchir* 1990;45:133–5.
- <sup>22</sup> Donnelly V, Foran A, Murphy J, McParland P, Keane D, O'Herlihy C. Neonatal brachial plexus palsy: an unpredictable injury. *Am J Obstet Gynecol* 2002;187:1209–12.
- <sup>23</sup> Walsh JM, Kandamany N, Ni Shuibhne N, Power H, Murphy JF, O'Herlihy C. Neonatal brachial plexus injury: comparison of incidence and antecedents between 2 decades. *Am J Obstet Gynecol* 2011;204:e1–e6.
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<sup>25</sup> Gonen R, Bader D, Ajami M. Effects of a policy of elective cesarean delivery in cases of suspected fetal macrosomia on the incidence of brachial plexus injury and the rate of cesarean delivery. *Am J Obstet Gynecol* 2000;183:1296–300.

<sup>26</sup> Kees S, Margalit V, Schiff E, Mashiah S, Carp HJ. Features of shoulder dystocia in a busy obstetric unit. *J Reprod Med* 2001;46:583–8.

<sup>27</sup> Bahar AM. Risk factors and fetal outcome in cases of shoulder dystocia compared with normal deliveries of a similar birthweight. *Br J Obstet Gynaecol* 1996;103:868–72.

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<sup>29</sup> Backe B, Magnussen EB, Johansen OJ, Sellaeg G, Russwurm H. Obstetric brachial plexus palsy: a birth injury not explained by the known risk factors. *Acta Obstet Gynecol Scand* 2008;87:1027–32.

<sup>30</sup> Tan KL. Brachial palsy. *J Obstet Gynaecol Br Commonw* 1973;80:60–2.

<sup>31</sup> Sjoberg I, Erichs K, Bjerre I. Cause and effect of obstetric (neonatal) brachial plexus palsy. *Acta Paediatr Scand* 1988;77:357–64.

<sup>32</sup> Gudmundsson S, Henningsson AC, Lindqvist P. Correlation of birth injury with maternal height and birthweight. *BJOG* 2005;112:764–7.

<sup>33</sup> Mollberg M, Hagberg H, Bager B, Lilja H, Ladfors L. Risk factors for obstetric brachial plexus palsy among neonates delivered by vacuum extraction. *Obstet Gynecol* 2005;106:913–8.

<sup>34</sup> Dawodu A, Sankaran-Kutty M, Rajan TV. Risk factors and prognosis for brachial plexus injury and clavicular fracture in neonates: a prospective analysis from the United Arab Emirates. *Ann Trop Paediatr* 1997;17:195–200.

<sup>35</sup> Olugbile A, Mascarenhas L. Review of shoulder dystocia at the Birmingham Women's Hospital. *J Obstet Gynaecol* 2000;20:267–70.

<sup>36</sup> Tandon S, Tandon V. Primiparity: a risk factor for brachial plexus injury in the presence of shoulder dystocia? *J Obstet Gynaecol* 2005;25:465–8.

<sup>37</sup> MacKenzie IZ, Shah M, Lean K, Dutton S, Newdick H, Tucker DE. Management of shoulder dystocia: trends in incidence and maternal and neonatal morbidity. *Obstet Gynecol* 2007;110:1059–68.

<sup>38</sup> Draycott TJ, Crofts JF, Ash JP, Wilson LV, Yard E, Sibanda T, et al. Improving neonatal outcome through practical shoulder dystocia training. *Obstet Gynecol* 2008;112:14–20.

<sup>39</sup> Melendez J, Bhatia R, Callis L, Woolf V, Yoong W. Severe shoulder dystocia leading to neonatal injury: a case control study. *Arch Gynecol Obstet* 2009;279:47–51.

<sup>40</sup> Evans-Jones G, Kay SP, Weindling AM, Cranny G, Ward A, Bradshaw A, et al. Congenital brachial palsy: incidence, causes, and outcome in the United Kingdom and Republic of Ireland. *Arch Dis Child Fetal Neonatal Ed* 2003;88:F185–9.

**Table 1–2.** Rate of Persistent Neonatal Brachial Plexus Palsy ↵

Authors	Publication date	Country	Study period	Total births	NBPP	NBPP follow-up more than 12 months	Persistent NBPP <sup>1</sup>	Rate of persistent NBPP <sup>2</sup>
Soni AL et al <sup>3</sup>	1985	Libya	1983	7,829	28	18	6	33%
Sjöberg I et al <sup>4</sup>	1984	Sweden	1973–1982	25,736	48	48	12	25%
Nocon JJ et al <sup>5</sup>	1993	United States	1986–1990	14,297	33	29	1	3%
Gherman RB et al <sup>6</sup>	1998	United States	1991–1995	58,565	48	45	4	9%
Gherman RB et al <sup>7</sup>	1998	United States	1995–1996	—	40	40	9	23%
Turrentine MA, Ramirez MM <sup>8</sup>	1999	United States	1996–1998	3,008	6	6	2	33%
Gonen R et al <sup>9</sup>	2000	Israel	1995–1999	16,416	17	11	1	9%
Donnelly V et al <sup>10</sup>	2002	Ireland	1994–1998	35,796	54	54	10	19%
Tandon S, Tandon V <sup>11</sup>	2005	United Kingdom	1989–1995	25,855	27	27	3	11%
Chauhan SP et al <sup>12</sup>	2005	United States	1980–2002	89,978	89	85	10	12%
Gurewitsch ED et al <sup>13</sup>	2006	United States	1993–2004	20,478	128	128	11	9%
Backe B et al <sup>14</sup>	2008	Norway	1991–2000	30,574	91	86	15	17%
Draycott TJ <sup>15</sup>	2008	United Kingdom	1996–1999;	39,220	30	30	8	27%
Rahman J et al <sup>16</sup>	2009	Saudi Arabia	1990–2005; 2001–2004		21	21	3	14%
Walsh JM et al <sup>17</sup>	2011	Ireland	1994–1998; 2004–2008	77,624	121	121	19	15%
<b>Total</b>				<b>445,376</b>	<b>781</b>	<b>749</b>	<b>114</b>	<b>15%</b>

Abbreviation: NBPP, neonatal brachial plexus palsy.

<sup>1</sup>NBPP lasting for at least 12 months.<sup>2</sup>Rate calculated from the number of NBPP cases monitored for at least 12 months.<sup>3</sup>Soni AL, Mir NA, Kishan J, Faquih AM, Elzouki AY. Brachial plexus injuries in babies born in hospital: an appraisal of risk factors in a developing country. *Ann Trop Paediatr* 1985;5:69–71.

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<sup>8</sup> Turrentine MA, Ramirez MM. Adverse perinatal events and subsequent cesarean rate. *Obstet Gynecol* 1999;94:185–8.

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<sup>10</sup> Donnelly V, Foran A, Murphy J, McParland P, Keane D, O'Herlihy C. Neonatal brachial plexus palsy: an unpredictable injury. *Am J Obstet Gynecol* 2002;187:1209–12.

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<sup>12</sup> Chauhan SP, Rose CH, Gherman RB, Magann EF, Holland MW, Morrison JC. Brachial plexus injury: a 23-year experience from a tertiary center. *Am J Obstet Gynecol* 2005;192:1795–800; discussion 1800–2.

<sup>13</sup> Gurewitsch ED, Johnson E, Hamzehzadeh S, Allen RH. Risk factors for brachial plexus injury with and without shoulder dystocia. *Am J Obstet Gynecol* 2006;194:486–92.

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<sup>15</sup> Draycott TJ, Crofts JF, Ash JP, Wilson LV, Yard E, Sibanda T, et al. Improving neonatal outcome through practical shoulder dystocia training. *Obstet Gynecol* 2008;112:14–20.

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<sup>17</sup> Walsh JM, Kandamany N, Ni Shuibhne N, Power H, Murphy JF, O'Herlihy C. Neonatal brachial plexus injury: comparison of incidence and antecedents between 2 decades. *Am J Obstet Gynecol* 2011;204:e1–e6.

**Table 1–3.** Frequency of Erb Palsy and Klumpke Palsy ↵

Authors	Country	Study period	Deliveries	Erb palsy	Rate (per 1,000 live births)	Klumpke palsy	Rate (per 1,000 live births)
Specht <sup>1</sup>	United States	1963–1972	19,314	11	0.6	0	0
Tan <sup>2</sup>	Singapore	1969–1971	90,436	57	0.6	0	0
Sjoberg <sup>3</sup>	Sweden	1973–1982	25,736	48	1.9	0	0
Acker <sup>4</sup>	United States	1975–1985	32,468	22	0.7	0	0
Jennett <sup>5</sup>	United States	1977–1990	57,597	36	0.6	0	0
Baskett <sup>6</sup>	Canada	1980–1989	40,518	33	0.8	0	0
Graham <sup>7</sup>	United States	1987–1991	14,358	15	1.0	0	0
Gherman <sup>8</sup>	United States	1995–1996	9,071	40	4.4	0	0
Jackson <sup>9</sup>	United States	1983–1996	8,258	21	2.5	1	0.1
Benjamin <sup>10</sup>	Saudi Arabia	1986–1987	2,222	10	4.5	1	0.5
Dawodu <sup>11</sup>	United Arab Emirates	1993–1995	9,231	27	2.9	1	0.1
Soni <sup>12</sup>	Libya	1983	7,829	28	3.6	12	1.5
Gordon <sup>13</sup>	United States	NM	31,700	60	1.9	3	0.1
Total			339,676	408	1.2	18	0.05

Abbreviation: NM, not measured.

<sup>1</sup> Specht EE. Brachial plexus palsy in the newborn. Incidence and prognosis. *Clin Orthop Relat Res* 1975;110:32–4.

<sup>2</sup> Tan KL. Brachial palsy. *J Obstet Gynaecol Br Commonw* 1973;80:60–2.

<sup>3</sup> Sjoberg I, Erichs K, Bjerre I. Cause and effect of obstetric (neonatal) brachial plexus palsy. *Acta Paediatr Scand* 1988;77:357–64.

<sup>4</sup> Acker DB, Gregory KD, Sachs BP, Friedman EA. Risk factors for Erb-Duchenne palsy. *Obstet Gynecol* 1988;71:389–92.

<sup>5</sup> Jennett RJ, Tarby TJ, Kreinick CJ. Brachial plexus palsy: an old problem revisited. *Am J Obstet Gynecol* 1992;166:1673–6; discussion 1676–7.

<sup>6</sup> Baskett TF, Allen AC. Perinatal implications of shoulder dystocia. *Obstet Gynecol* 1995;86:14–7.

<sup>7</sup> Graham EM, Forouzan I, Morgan MA. A retrospective analysis of Erb's palsy cases and their relation to birth weight and trauma at delivery. *J Matern Fetal Med* 1997;6:1–5.

<sup>8</sup> Gherman RB, Ouzounian JG, Miller DA, Kwok L, Goodwin TM. Spontaneous vaginal delivery: a risk factor for Erb's palsy? *Am J Obstet Gynecol* 1998;178:423–7.

<sup>9</sup> Jackson ST, Hoffer MM, Parrish N. Brachial-plexus palsy in the newborn. *J Bone Joint Surg Am* 1988;70:1217–20.

<sup>10</sup> Benjamin B, Khan MR. Pattern of external birth trauma in southwestern Saudi Arabia. *J Trauma* 1993;35:737–41.

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<sup>12</sup> Soni AL, Mir NA, Kishan J, Faquih AM, Elzouki AY. Brachial plexus injuries in babies born in hospital: an appraisal of risk factors in a developing country. *Ann Trop Paediatr* 1985;5:69–71.

<sup>13</sup> Gordon M, Rich H, Deutschberger J, Green M. The immediate and long-term outcome of obstetric birth trauma. I. Brachial plexus paralysis. *Am J Obstet Gynecol* 1973;117:51–6.

**Table 1–4.** Neonatal Brachial Plexus Palsy Without Concomitant Shoulder Dystocia ↵

Authors	Publication date	Country	Study period	Total births	NBPP	Number of cases of NBPP without SD	NBPP without SD (%)
Salonen IS et al <sup>1</sup>	1990	Finland	1981–1987	14,265	16	8	50%
Jennett RJ <sup>2</sup>	1992	United States	1977–1990	57,597	39	22	56%
Nocon JJ et al <sup>3</sup>	1993	United States	1986–1990	14,297	33	5	15%
Ouzounian JG et al <sup>4</sup>	1998	United States	—	—	63	4	6%
Donnelly V et al <sup>5</sup>	2002	Ireland	1994–1998	35,796	54	7	13%
Tandon S, Tandon V <sup>6</sup>	2005	United Kingdom	1989–1995	25,855	27	4	15%
Gudmundsson S et al <sup>7</sup>	2005	Sweden	1990–1996	16,743	51	32	63%
Chauhan SP et al <sup>8</sup>	2005	United States	1980–2002	89,978	89	39	44%
Mollberg M et al <sup>9</sup>	2005	Sweden	1987–1997	1,213,987	2,399	1,166	49%
Gurewitsch ED et al <sup>10</sup>	2006	United States	1993–2004	20,478	128	45	35%
Backe B et al <sup>11</sup>	2008	Norway	1991–2000	30,574	91	74	81%
Walsh JM et al <sup>12</sup>	2011	Ireland	2004–2008; 1994–1998	77,624	121	39	32%
Total				1,597,194	3,111	1,445	46%

Abbreviations: NBPP, neonatal brachial plexus palsy; SD, shoulder dystocia.

<sup>1</sup> Salonen IS, Uusitalo R. Birth injuries: incidence and predisposing factors. *Z Kinderchir* 1990;45:133–5.

<sup>2</sup> Jennett RJ, Tarby TJ, Kreinick CJ. Brachial plexus palsy: an old problem revisited. *Am J Obstet Gynecol* 1992;166:1673–6; discussion 1676–7.

<sup>3</sup> Nocon JJ, McKenzie DK, Thomas LJ, Hansell RS. Shoulder dystocia: an analysis of risks and obstetric maneuvers. *Am J Obstet Gynecol* 1993;168:1732–7; discussion 1737–9.

<sup>4</sup> Ouzounian JG, Korst LM, Phelan JP. Permanent Erb's palsy: a lack of a relationship with obstetrical risk factors [published erratum appears in Am J Perinatol 2003;20:275]. *Am J Perinatol* 1998;15:221–3.

<sup>5</sup> Donnelly V, Foran A, Murphy J, McParland P, Keane D, O'Herlihy C. Neonatal brachial plexus palsy: an unpredictable injury. *Am J Obstet Gynecol* 2002;187:1209–12.

<sup>6</sup> Tandon S, Tandon V. Primiparity: a risk factor for brachial plexus injury in the presence of shoulder dystocia? *J Obstet Gynaecol* 2005;25:465–8.

<sup>7</sup> Gudmundsson S, Henningsson AC, Lindqvist P. Correlation of birth injury with maternal height and birthweight. *BJOG* 2005;112:764–7.

<sup>8</sup> Chauhan SP, Rose CH, Gherman RB, Magann EF, Holland MW, Morrison JC. Brachial plexus injury: a 23-year experience from a tertiary center. *Am J Obstet Gynecol* 2005;192:1795–800; discussion 1800–2.

<sup>9</sup> Mollberg M, Hagberg H, Bager B, Lilja H, Ladfors L. Risk factors for obstetric brachial plexus palsy among neonates delivered by vacuum extraction. *Obstet Gynecol* 2005;106:913–8.

<sup>10</sup> Gurewitsch ED, Johnson E, Hamzehzadeh S, Allen RH. Risk factors for brachial plexus injury with and without shoulder dystocia. *Am J Obstet Gynecol* 2006;194:486–92.

<sup>11</sup> Backe B, Magnussen EB, Johansen OJ, Sellaeg G, Russwurm H. Obstetric brachial plexus palsy: a birth injury not explained by the known risk factors. *Acta Obstet Gynecol Scand* 2008;87:1027–32.

<sup>12</sup> Walsh JM, Kandamany N, Ni Shuibhne N, Power H, Murphy JF, O'Herlihy C. Neonatal brachial plexus injury: comparison of incidence and antecedents between 2 decades. *Am J Obstet Gynecol* 2011;204:324.e1–e6.

**Table 1–5. Cesarean Delivery and Neonatal Brachial Plexus Palsy** ↵

Authors	Publication date	Country	Study period	Number of cesarean deliveries	Cesarean delivery with NBPP	Rate of cesarean delivery with NBPP (per 1,000 births)
Tan KL <sup>1</sup>	1973	Singapore	1969–1971	5,023	4	0.8
Graham EM et al <sup>2</sup>	1997	United States	1987–1991	2,874	1	0.3
Gherman RB et al <sup>3</sup>	1997	United States	1991–1995	8,451	6	0.7
Gilbert WM et al <sup>4</sup>	1999	United States	1994–1995	216,414	60	0.3
Gurewitsch ED et al <sup>5</sup>	2006	United States	1993–2004	4,587	7	1.5
Walsh JM et al <sup>6</sup>	2011	Ireland	1994–1998; 2004–2008	11,526	5	0.4
Total				248,875	83	0.3

Abbreviation: NBPP, neonatal brachial plexus palsy.

<sup>1</sup>Tan KL. Brachial palsy. *J Obstet Gynaecol Br Commonw* 1973;80:60–2.

<sup>2</sup>Graham EM, Forouzan I, Morgan MA. A retrospective analysis of Erb's palsy cases and their relation to birth weight and trauma at delivery. *J Matern Fetal Med* 1997;6:1–5.

<sup>3</sup>Gherman RB, Goodwin TM, Ouzounian JG, Miller DA, Paul RH. Brachial plexus palsy associated with cesarean section: an in utero injury? *Am J Obstet Gynecol* 1997;177:1162–4.

<sup>4</sup>Gilbert WM, Nesbitt TS, Danielsen B. Associated factors in 1611 cases of brachial plexus injury. *Obstet Gynecol* 1999;93:536–40.

<sup>5</sup>Gurewitsch ED, Johnson E, Hamzehzadeh S, Allen RH. Risk factors for brachial plexus injury with and without shoulder dystocia. *Am J Obstet Gynecol* 2006;194:486–92.

<sup>6</sup>Walsh JM, Kandamany N, Ni Shuibhne N, Power H, Murphy JF, O'Herlihy C. Neonatal brachial plexus injury: comparison of incidence and antecedents between 2 decades. *Am J Obstet Gynecol* 2011;204:e1–e6.

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# Chapter 2

## Risk and Predictive Factors

### Reliability of Risk Factors as Predictors for Neonatal Brachial Plexus Palsy

Various risk factors have been described in association with neonatal brachial plexus palsy (NBPP). Overall, however, these risk factors have not been shown to be statistically reliable or clinically useful predictors for the occurrence of NBPP.

Fetal macrosomia, either alone or in conjunction with maternal diabetes, has been reported as a risk factor for NBPP. A reasonable threshold for the diagnosis of macrosomia is a birth weight of 4,000 g or more (1). As birth weight increases, NBPP occurs more frequently, but most NBPP cases occur in mothers who do not have diabetes and in neonates who weigh less than 4,000 g. This finding has been corroborated in at least seven studies over a 20-year period, compiled in Table 2-1 (2–9). In another study, Lipscomb and colleagues evaluated 157 infants with birth weight greater than 4,500 g and demonstrated no persistent injuries in seven cases of NBPP (10). Ecker and colleagues also demonstrated that NBPP occurs more frequently with increasing birth weight (11). However, that study also showed that suspected macrosomia in and of

itself as an indication for cesarean delivery would have a limited effect in reducing the incidence of NBPP. In fact, even in women with diabetes with an estimated fetal weight greater than 4,500 g, the positive predictive value for NBPP was only 5%. In mothers who did not have diabetes, it was less than 2%. Pondaag and colleagues demonstrated that increasing birth weight correlated with higher rates of NBPP and higher rates of permanent injury, but conceded that widespread prophylactic cesarean delivery to prevent NBPP was not a practical strategy (12).

Even when ultrasonography is used, clinical estimates of birth weight are only within 15–20% of actual birth weight 95% of the time (1). King and colleagues recently demonstrated that ultrasonography only had a 40% sensitivity for detecting birth weight greater than 4,500 g, and abdominal palpation had a sensitivity of 15% (13). Few studies have analyzed the positive predictive value or odds ratios (ORs) of estimated fetal weight and NBPP. In the few case-control studies from which data can be extrapolated, the positive predictive value for NBPP of a birth weight greater than 4,500 g in the setting of delivery complicated by clinical shoulder dystocia ranges from 12% to 40%. Other studies have

**Table 2–1.** Various Risk Factors and Their Association With Neonatal Brachial Plexus Palsy ↵

Authors	Number of cases with birth weight greater than 4,000 g (%)	Number of cases with birth weight greater than 4,500 g (%)	Number of cases with diabetes (%)	Number of cases with shoulder dystocia (%)	Number of cases with NBPP		
	Total	Transient NBPP (%)	Permanent NBPP (%)				
Nocon 1993 <sup>1</sup>	NM	NM	11 (33.3)	28 (84.8)	33	32 (97.0)	1 (3.0)
Graham 1997 <sup>2</sup>	6 (42.8)	2 (14.3)	NM	8 (57.1)	14	NM	NM
Ouzounian 1998 <sup>3</sup>	51 (80.9)	26 (41.0)	7 (11.7)	59 (93.6)	63	0	63
Gherman 1998 <sup>4</sup>	21 (52.5)	9 (22.5)	9 (22.5)	17 (42.5)	40	33 (82.5)	7 (17.5)
Gherman 2003 <sup>5</sup>	44 (36.9)	27 (22.7)	22 (18.5)	98 (82.3)	119	66 (55.5)	53 (44.5)
Poggi 2003 <sup>6,7</sup>	49 (36.8)	17 (12.8)	11 (8.3)	127 (95.5)	133	0	133
Chauhan 2005 <sup>8</sup>	31 (36.4)	NM	10 (11.7)	46 (54.1)	85	75 (88.2)	10 (11.8)
Ouzounian 2012 <sup>9,10</sup>	22 (52.4)	8 (19.0)	10 (23.8)	42 (100)	42	NM	NM

Abbreviations: NBPP, neonatal brachial plexus palsy; NM, not measured.

<sup>1</sup> Nocon JJ, McKenzie DK, Thomas LJ, Hansell RS. Shoulder dystocia: an analysis of risks and obstetric maneuvers. *Am J Obstet Gynecol* 1993;168:1732–7; discussion 1737–9.

<sup>2</sup> Graham EM, Forouzan I, Morgan MA. A retrospective analysis of Erb's palsy cases and their relation to birth weight and trauma at delivery. *J Matern Fetal Med* 1997;6:1–5.

<sup>3</sup> Ouzounian JG, Gherman RB. Shoulder dystocia: are historic risk factors reliable predictors? *Am J Obstet Gynecol* 2005;192:1933–5; discussion 1935–8.

<sup>4</sup> Gherman RB, Ouzounian JG, Miller DA, Kwok L, Goodwin TM. Spontaneous vaginal delivery: a risk factor for Erb's palsy? *Am J Obstet Gynecol* 1998;178:423–7.

<sup>5</sup> Gherman RB, Ouzounian JG, Satin AJ, Goodwin TM, Phelan JP. A comparison of shoulder dystocia-associated transient and permanent brachial plexus palsies. *Obstet Gynecol* 2003;102:544–8.

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<sup>7</sup> Transient injuries were excluded from analysis.

<sup>8</sup> Chauhan SP, Rose CH, Gherman RB, Magann EF, Holland MW, Morrison JC. Brachial plexus injury: a 23-year experience from a tertiary center. *Am J Obstet Gynecol* 2005;192:1795–800; discussion 1800–2.

<sup>9</sup> Ouzounian JG, Korst LM, Miller DA, Lee RH. Brachial plexus palsy and shoulder dystocia: obstetric risk factors remain elusive. *Am J Perinatol* 2013;30:303–7.

<sup>10</sup> Study limited to shoulder dystocia cases only.

demonstrated various cumulative ORs for the typical risk factors analyzed (14). Thus, such estimates are of limited utility for contemporaneous clinical management. Furthermore, no data exist to support the claim that estimated fetal weight can be used prophylactically to reduce the incidence of NBPP.

Many cases of NBPP occur in conjunction with recognized shoulder dystocia. In fact, many of the obstetric risk factors cited in conjunction with NBPP are reported to be associated with clinically evident shoulder dystocia. The same biomechanical factors that predispose a fetus to develop NBPP (forces of labor, in utero malpositioning, or failure of truncal rotation) also may predispose a fetus to shoulder dystocia. The presence of both recognized shoulder dystocia and NBPP can lead to an erroneous retrospective inference of causation. However, as previously discussed, studies have demonstrated that the occurrence of NBPP in a delivery complicated by shoulder dystocia has an incidence ranging from 4% to 23% (2, 9, 12–23). Similar to cases of NBPP, the majority of clinically diagnosed shoulder dystocia cases occur in infants with a birth weight less than 4,500 g and without maternal diabetes (see Table 2-2). When studied alone or with NBPP, risk factors for shoulder dystocia are not reliable predictors for its occurrence. For example, in a 2005 study of more than 1,600 cases of shoulder dystocia, the clinical triad of birth weight greater than 4,500 g, oxytocin use, and labor induction had a positive predictive value for shoulder dystocia of only 3.4% (15). In cases complicated by shoulder dystocia, higher birth weight can also lead to an increased incidence of NBPP (12). Additionally, in women with recurrent shoulder dystocia, the risk of NBPP can be as high as 4.5%, compared with 1–2% with the first episode of shoulder dystocia (24).

The application of fundal pressure during a delivery in which shoulder dystocia is recognized can exacerbate shoulder impaction and lead to an increased risk of NBPP. This finding has been demonstrated in at least one study (25). The relation-

ship, if any, of fundal pressure to the occurrence of NBPP in deliveries without shoulder dystocia is unknown.

Clinician-applied traction and lateral bending of the fetal neck have been implicated as causative factors in some cases of NBPP. However, NBPP also has been shown to occur entirely unrelated to traction, with studies demonstrating cases of both transient and persistent NBPP in fetuses delivered vaginally without clinically evident shoulder dystocia or fetuses delivered by cesarean without shoulder dystocia (5, 26–28). Other studies have reported NBPP occurring in the posterior shoulder in the presence of a delivery complicated by anterior shoulder impaction (29, 30).

Various risk factors have been described in association with NBPP. They include fetal malposition, labor induction, labor abnormalities, operative vaginal delivery, fetal macrosomia, and shoulder dystocia. Several studies of these clinical factors are summarized in Table 2-3. Overall, except for shoulder dystocia, these risk factors have not been shown to be statistically significant or clinically useful predictors for the occurrence of NBPP (9).

## Association of Risk and Outcome Versus Prediction of Outcome

Although multiple clinical risk factors have been described in association with NBPP, it is important to explore whether these associations have been demonstrated to be useful or effective in accurately predicting this outcome. The strength of an association is assessed by measures such as relative risk or ORs. If there is no association between a factor and outcome, that factor will not be useful in prediction. However, even if strong associations exist between a given factor (eg, a rapid second stage of labor) and a given outcome (eg, clinically evident shoulder dystocia), such a factor, or combination of factors, cannot necessarily be used for accurate prediction (31, 32).

**Table 2–2.** Various Risk Factors and Their Association With Shoulder Dystocia ↵

Authors	Number of patients	Number of cases of shoulder dystocia	Number of cases of birth weight greater than 4,000 g (%)	Number of cases of birth weight greater than 4,500 g	Number of cases with diabetes (%)	Number of cases of NBPP (%)
Ouzounian 2005 <sup>1</sup>	267,228	1,686	946 (56.1)	326 (19.3)	250 (14.8)	NM
Gherman 1997 <sup>2</sup>	44,072	236	99 (41.9)	38 (16.1)	90 (36.0)	37 (16.2)
Ouzounian 2012 <sup>3</sup>	16,071	221	109 (49.3)	32 (14.5)	45 (20.4)	42 (19.0)
Nocon 1993 <sup>4</sup>	12,532	185	NM	NM	11 (5.9)	28 (15.1)
Baskett 1995 <sup>5</sup>	40,518	254	158 (62.2)	69 (27.1)	NM	33 (13.0)
Hopwood 1982 <sup>6</sup>	11,883	92	NM	NM	5 (5.4)	4 (4.3)
Geary 1995 <sup>7</sup>	10,486	66	58 (87.9)	23 (34.8)	2 (3.0)	8 (12.1)
Kees 2001 <sup>8</sup>	24,000	56	20 (35.7)	NM	6 (10.7)	13 (23.2)
Lewis 1998 <sup>9</sup>	1,622	99	35 (35.0)	8 (8.0)	5 (5.0)	NM
Sandmire 1986 <sup>10</sup>	14,806	73	1,755 (11.8)	523 (3.5)	6 (8.2)	12 (16.4)

Abbreviations: NBPP, neonatal brachial plexus palsy; NM, not measured.

<sup>1</sup> Ouzounian JG, Gherman RB: Shoulder dystocia: Are historic risk factors reliable predictors? *Am J Obstet Gynecol* 2005;192:1933–8.

<sup>2</sup> Gherman RB, Goodwin TM, Souter I, Neumann K, Ouzounian JG, Paul RH. The McRoberts' maneuver for the alleviation of shoulder dystocia: how successful is it? *Am J Obstet Gynecol* 1997;176:656–61.

<sup>3</sup> Ouzounian JG, Korst LM, Miller DA, Lee RH. Brachial plexus palsy and shoulder dystocia: obstetric risk factors remain elusive. *Am J Perinatol* 2013;30:303–7.

<sup>4</sup> Nocon JJ, McKenzie DK, Thomas LJ, Hansell RS. Shoulder dystocia: an analysis of risks and obstetric maneuvers. *Am J Obstet Gynecol* 1993;168:1732–7; discussion 1737–9.

<sup>5</sup> Baskett TF, Allen AC. Perinatal implications of shoulder dystocia. *Obstet Gynecol* 1995;86:14–7.

<sup>6</sup> Hopwood HG Jr. Shoulder dystocia: fifteen years' experience in a community hospital. *Am J Obstet Gynecol* 1982;144:162–6.

<sup>7</sup> Geary M, McParland P, Johnson H, Stronge J. Shoulder dystocia—is it predictable? *Eur J Obstet Gynecol Reprod Biol* 1995;62:15–8.

<sup>8</sup> Kees S, Margalit V, Schiff E, Mashiach S, Carp HJ. Features of shoulder dystocia in a busy obstetric unit. *J Reprod Med* 2001;46:583–8.

<sup>9</sup> Lewis DF, Edwards MS, Asrat T, Adair CD, Brooks G, London S. Can shoulder dystocia be predicted? Preconceptive and prenatal factors. *J Reprod Med* 1998;43:654–8.

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**Table 2–3.** Other Risk Factors for Neonatal Brachial Plexus Palsy ↵

Authors	Number of cases of NBPP	Number of cases of second stage of labor longer than 2 h (%)	Number of cases of second stage of labor less than 20 min (%)	Number of cases of vacuum or forceps used (%)	Number of cases of oxytocin use (%)	Number of cases of labor induction (%)
Ouzounian 1997 <sup>1</sup>	4	2 (50.0)	0	1 (25.0%)	NM	1 (25.0)
Ouzounian 1998 <sup>2</sup>	63	9 (14.3)	NM	13 (20.6)	NM	NM
Gherman 1998 <sup>3</sup>	40	8 (20.0)	15 (37.5)	3 (7.5)	18 (45.0)	NM
Poggi 2003 <sup>4</sup>	74	7 (9.5)	21 (28.0)	22 (12.5)	NM	NM
Gherman 2003 <sup>5</sup>	98	21 (21.4)	12 (12.2)	21 (21.4)	59 (60.2)	NM
Chauhan 2005 <sup>6</sup>	85	NM	NM	NM	34 (40.0)	9 (10.6)
Mean		16.8%	22.2%	21.5%	42.1%	11.2%

Abbreviation: NBPP, neonatal brachial plexus palsy; NM, not measured.

<sup>1</sup> Ouzounian JG, Korst LM, Phelan JP. Permanent Erb palsy: a traction-related injury? *Obstet Gynecol* 1997;89:139–41.

<sup>2</sup> Ouzounian JG, Gherman RB. Shoulder dystocia: are historic risk factors reliable predictors? *Am J Obstet Gynecol* 2005;192:1933–5; discussion 1935–8.

<sup>3</sup> Gherman RB, Ouzounian JG, Miller DA, Kwok L, Goodwin TM. Spontaneous vaginal delivery: a risk factor for Erb's palsy? *Am J Obstet Gynecol* 1998;178:423–7.

<sup>4</sup> Poggi SH, Stallings SP, Ghidini A, Spong CY, Deering SH, Allen RH. Intrapartum risk factors for permanent brachial plexus injury. *Am J Obstet Gynecol* 2003;189:725–9.

<sup>5</sup> Gherman RB, Ouzounian JG, Satin AJ, Goodwin TM, Phelan JP. A comparison of shoulder dystocia-associated transient and permanent brachial plexus palsies. *Obstet Gynecol* 2003;102:544–8.

<sup>6</sup> Chauhan SP, Rose CH, Gherman RB, Magann EF, Holland MW, Morrison JC. Brachial plexus injury: a 23-year experience from a tertiary center. *Am J Obstet Gynecol* 2005;192:1795–800; discussion 1800–2.

Pepe and colleagues have described why measures such as ORs do not provide adequate insight into predictive capability (32). The capability of a predictive model is based on its ability to correctly identify those who will have an outcome (ie, the true-positive fraction, or sensitivity) as well as correctly identify those who will not have an outcome (ie, the true-negative fraction, or specificity). The inability of measures such as ORs to provide good insight into prediction may be clarified by specific examples. As Pepe and colleagues point out, a risk factor with an OR of 3, which some would consider evidence of a moderately strong association, is quite poor at classifying (or predicting) whether an individual will have a certain outcome. A specific numeric example might make this concept clearer.

**Table 2–4** corresponds to the situation in which a complication occurs in 1% of a population, and the strength of association between that complication and a certain risk factor is represented by an OR of 2.7. In this case, the OR translates to a sensitivity of approximately 80% and a specificity of approximately 40%. As the table demonstrates, although 80% of women who experience the complication will be identified by the risk factor, 60% of women who will be complication-free will be wrongly predicted to experience the complication. From another perspective, it is more than 70 times more likely that a woman with the risk factor will not have a complication. (For more information on odds ratios and sensitivity and specificity, please see **Tables 2–5 and 2–6**.)

**Table 2–4.** Sample Calculation of Risk of Complications\*

		Complication		
		Present	Absent	
Risk factor	Present	80	5,940	
	Absent	20	3,960	
		100	9,900	

\*It is assumed that 100 of 10,000 women experience a complication. If the odds ratio for the association between a risk factor and the complication is 2.7 (equivalent to a sensitivity of 80% in conjunction with a specificity of 40%), 80 women who have the complication will have the relevant risk factor as well. These 80 women are the “true-positives.” Conversely, of the 9,900 women without the complication, 3,960 will not have the risk factor (ie, “true-negatives”). Thus, 5,940 (60%) will have the risk factor and will be wrongly predicted to incur the complication. By dividing the 5,940 who have the risk factor but experience no complication by the 80 who have the risk factor and experience the complication, the result is 74.25, meaning that it is more than 70 times more likely that women with the risk factor will not experience the condition.

**Table 2–5.** Odds Ratio Calculation\*

Disease presence	Exposed	Unexposed	
Disease	a	b	
No disease	c	d	

\*The exposure odds ratio (OR) for a set of case-control data is the ratio of the odds in favor of exposure among the cases (a/b) to the odds in favor of exposure among noncases (c/d). A 2x2 table can be used to illustrate this calculation of OR. In this example, the OR is ad/bc.

Porta MS, Greenland S, Last JM, editors. A dictionary of epidemiology. Edited for the international epidemiological association. 5th ed. New York (NY): Oxford University Press; 2008.

**Table 2–6.** Sensitivity and Specificity Calculation\*

Test result	True status		Total
	Diseased	Not diseased	
Positive	a	b	a + b
Negative	c	d	c + d
Total	a + c	b + d	a + b + c + d

a=diseased individuals detected by the test (true-positives)

b=nondiseased individuals positive by the test (false-positives)

c=diseased individuals not detected by the test (false-negatives)

d=nondiseased individuals negative by the test (true-negatives)

\*Sensitivity is the proportion of truly diseased persons in the screened population who are identified as diseased by the screening test. Specificity is the proportion of truly nondiseased persons who are so identified by the screening test. Sensitivity=a/(a+c); specificity=d/(b+d).

Porta MS, Greenland S, Last JM, editors. A dictionary of epidemiology. Edited for the international epidemiological association. 5th ed. New York (NY): Oxford University Press; 2008.

## Application to Practice

Notwithstanding the unreliability of specific risk factors to predict NBPP or clinically apparent shoulder dystocia in a specific case, there are three clinical situations of concern to practitioners in which an alteration of usual obstetric management might be considered to reduce the risk of shoulder dystocia and brachial plexus injury, 1) suspected fetal macrosomia with estimated fetal weight exceeding 5,000 g in a woman without diabetes or 4,500 g in a woman with diabetes; 2) prior recognized shoulder dystocia, especially with a severe neonatal injury; and 3) midpelvic operative vaginal delivery with fetal birth weight more than 4,000 g. Even in these circumstances, the occurrence of NBPP is relatively low, and with proper informed consent, numerous clinical situations exist in which these risk factors alone should not dictate a particular course of management.

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# Chapter 3

## Pathophysiology and Causation ↵

The descent and emergence of the fetus from the uterus and pelvis is a result of uterine contractions and maternal pushing or expulsive forces and, when required, pulling or traction (tension) forces applied by a birth attendant. The application of these forces—internal (endogenous) and external (exogenous)—during labor and delivery is a biomechanical process. Significant endogenous forces are generated through natural physical events to move a fetus from the uterus through the birth canal and out of the maternal pelvis. Exogenous forces also can be significant. Injury to a nerve is similarly a biomechanical phenomenon. Forces applied to a nerve in the course of labor and delivery can damage nerve tissue, resulting in functional and structural deficits. Accordingly, biomechanics is a means through which the causes of neonatal brachial plexus palsy (NBPP) have been and continue to be investigated and understood.

This section addresses, from a biomechanical perspective, the relationship of endogenous and exogenous forces to transient and persistent NBPP. Stretch in the brachial plexus occurs during the birth process itself, as shown by both computer and physical models (1, 2), and it can occur in the nerves of either or both anterior and poste-

rior shoulders. This stretch results from differential motion between the fetal head and shoulders after some element of the maternal anatomy halts or retards the progression of the larger shoulders through the birth canal (1, 3).

It is generally acknowledged in the clinical literature that there can be wide variation in the biomechanical response of fetus and neonate—a precept long recognized in the biomechanical engineering and injury biomechanics communities. Whether as a result of variation in fetal well-being (as indicated by acid-base status), size, environmental effects, tone of the surrounding neck muscles, or other factors, some fetuses are more or less susceptible to injury than others (4). In addition, the biomechanics of delivery in relation to maternal anatomy and physiology also vary greatly. Thus, although it is impossible to directly compare one delivery with another, the general trends in forces and responses associated with a particular injury can be understood.

### Biomechanics of the Delivery Process

Both uterine contractions and maternal pushing efforts produce pressure within the uterus. This pressure produces a force that moves the fetus

into and through the pelvis. Force is measured either in Newtons (N) or pounds of force (lbf); the approximate conversion is 4 Newtons per pound. These maternally derived forces also are referred to as endogenous or expulsive forces. Clinician-applied forces are referred to as exogenous or extraction forces. Both of these types of forces (endogenous and exogenous) have a direct effect on the fetus as a whole and on various discrete anatomic structures. Maternal forces alone are an accepted cause of at least transient NBPP by most investigators (5). Brachial plexus stretch can also be increased by traction applied by the birth attendant—typically described as a downward lateral traction, with bending of the fetus' neck away from the anterior shoulder (ie, toward the posterior shoulder or floor) (1, 6). However, in the presence of shoulder dystocia, even properly applied axial traction will necessarily increase stretch of the brachial plexus.

There are two major components to the forces of the labor and delivery process: 1) compression and 2) traction. Compression can be conceptualized as a pushing force on an object, whereas traction is a pulling force. In the context of delivery, axial traction is traction applied in alignment with the fetal cervicothoracic spine (Fig. 3-1; see color plate). Axial traction is distinct from horizontal traction—traction within a plane parallel to the delivery table. Traction applied in the plane of the fetal cervicothoracic spine is typically along a vector estimated to be 25–45 degrees below the horizontal plane when the woman in labor is in a lithotomy position. Thus, although axial traction also is downward, it is applied without lateral bending of the fetal neck (ie, without bending the neck toward the floor or the ceiling).

In examining the biomechanics of labor and its effects on the fetus, it is important to differentiate between the force that is acting on the fetus' body as a whole and those forces that may be transmitted differentially to discrete anatomic structures. For example, uterine contractions result in a compression force to the fetus that acts to move the entire fetus down the birth canal. If, during this movement, a structure obstructs a body

part while another body segment continues moving forward, the difference in motion can result in either a pulling force on the tissues that connect the two regions or a bending force on a rigid, bony body part. Similarly, traction applied by the clinician results in a pulling force transmitted to the head and neck that is then transferred to the shoulders. However, if the anterior fetal shoulder becomes impacted behind the symphysis pubis, the traction applied to the fetus can be transmitted as both a traction and a compression force to soft tissues and other structures (eg, nerves) in the region of impaction. Thus, the effects of applied forces on the fetus' body are complex and may be in a different direction and of a different type than would be initially anticipated based on the applied force alone.

When a part of the body is subjected to a force, it will deform—either being stretched or compressed. The amount of deformation that a structure experiences is a key measure to estimate whether it will be damaged—either through fracture (bone) or rupture (nerve). Deformation is typically quantified by *strain*, which is defined as the change in the length of the structure normalized for its original length. Strain is given as the percent change in the measured dimension. In lay terms, a traction-based strain is commonly referred to as stretch. If a spring is pulled, then “100% stretch” indicates that it has increased in length by 100% of its original size (ie, doubled in length). Conversely, if the same spring is compacted, then “25% compression” indicates that the structure has decreased in length to 75% of its original size. When predicting whether delivery forces are likely to cause an injury, both the amount of deformation and susceptibility to injury should be considered.

Keeping in mind the general biomechanics of delivery, one must consider the specific forces at work during labor and how those forces can injure the brachial plexus. The question is often posed as to whether clinician-applied traction used in a particular delivery is “normal” or “abnormal.” There are two challenges in making such an assessment: 1) quantifying the applied force for a clinical delivery and 2) determining what is normal. The role

of endogenous and exogenous forces in labor and delivery has been studied extensively. Because investigation of the biomechanics of neonatal injury in humans is not possible, several models have been used to study the pathophysiology of NBPP. These include physical, cadaveric, animal, and computer models.

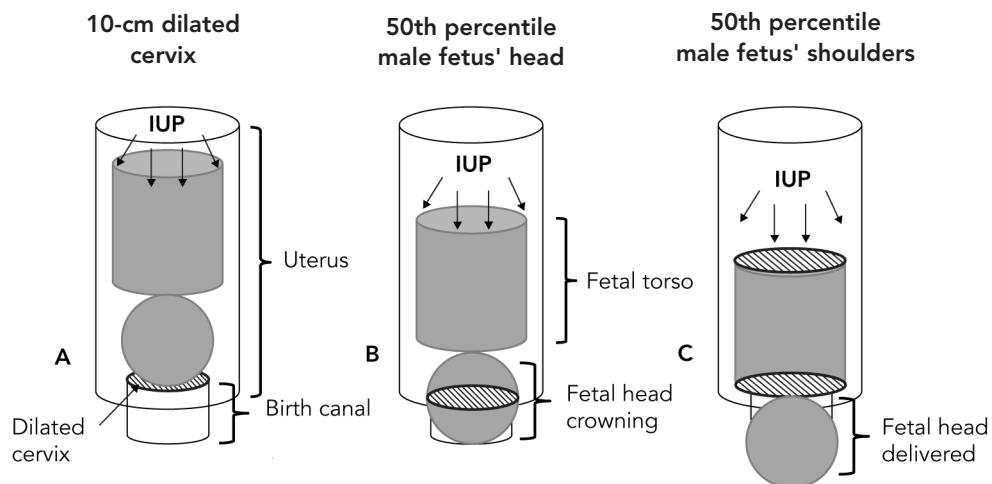
## Endogenous Forces

Maternal expulsive forces result from an increase in intrauterine pressure created by a combination of uterine contractions and an increase in abdominal pressure through the Valsalva maneuver. To calculate maternal expulsive forces, multiply the pressure by the area over which the pressure is applied. Various models have been used to determine the area over which intrauterine force is acting.

Using a piston as the simplest representation of the expulsion of the fetus from the uterus, the area used to calculate force is based on the cross-sectional area of the piston (Fig. 3-2). The 10-cm diameter of a completely dilated cervix has been used to represent this cross-sectional area (Fig. 3-2A) (5).

Using a measured intrauterine pressure of 80 mm Hg (10.3 kPa) in that single case study, investigators estimated a peak force of approximately 84 N (see Box 3-1).

It is readily apparent that the cervix must stretch beyond 10 cm to accommodate the delivery of a term fetus because even the head of a 50th percentile male fetus has a greater cross-sectional area than the  $78.5 \text{ cm}^2$  of the fully dilated cervix (Fig. 3-2B) (7, 8). A broader set of calculations based on anthropometric measurements of the fetal head demonstrated that maternal forces, the combination of uterine contractions and maternal pushing, are likely to be at least 140–160 N during the second stage of labor when intrauterine-pressure of 120 mm Hg is common (8). Opinions diverge as to whether the obstructing area of the piston is better represented by the fetus' body, which has an even larger cross-sectional area than the head (Fig. 3-2C) and, therefore, higher resulting forces from the same intrauterine pressure (8). Thus, it can be anticipated that maternal forces during the second stage of labor will reach at least 140 N with an average-sized fetus.



**Figure 3-2.** This simple representation of the expulsion of the fetus from the uterus demonstrates how intrauterine pressure can be used to calculate the effective endogenous force based on the cross-sectional area of (A) a 10-cm dilated cervix 84 N at 80 mm Hg; 126 N at 120 mm Hg, (B) the cross-sectional area of a 50th percentile male fetus' head 92 N at 80 mm Hg; 138 N at 120 mm Hg, and (C) the cross-sectional area of a 50th percentile male fetus' shoulders 111 N at 80 mm Hg; 167 N at 120 mm Hg. Abbreviation: IUP, intrauterine pressure. ↵

### Box 3-1. Calculation of Endogenous (Maternal) Delivery Forces ↵

Uterine contractions generate a force that moves the fetus down the birth canal during labor. While this is a complex loading environment, involving both direct contact between the endometrium/myometrium and the fetus as well as the development of pressure in the amniotic fluid, a reasonable estimate of the force developed can be calculated based on the following equation:

$$F = P \times A$$

Where F is the resulting maternal force, P is the intrauterine pressure developed during contractions and pushing efforts, and A is the cross-sectional area over which that pressure is applied.

Intrauterine pressure has been measured scientifically to range between 55 mm Hg and 120 mm Hg (1). Because of the complex cross-sectional area of a fetus, the specific area that should be used has been debated. Estimates of the maternal force can be made using various cross-sectional areas and pressures. Three different sample areas have been modeled in Figure 3-2:

- A. A circular, fully dilated cervix with a diameter of 10 cm
- B. An elliptical head of a 50th percentile male neonate (2)
  - Circumference of 35.8 cm
  - Minor axis (biparietal diameter = 7.4 cm) set at half of the major axis (frontal-occipital diameter = 14.8 cm)
- C. An elliptical torso of a 50th percentile male neonate (3)
  - Bisacromial diameter = 15.8 cm
  - Anterior-posterior chest thickness = 8.4 cm

Model	Cross-Sectional Area		Maternal Force 80 mm Hg		Maternal Force 120 mm Hg	
			N	lbf	N	lbf
A. Circular cervix	80 cm <sup>2</sup>		83.7 N	18.8 lbf	125.6 N	28.2 lbf
B. Elliptical head	86 cm <sup>2</sup>		91.8 N	20.6 lbf	137.6 N	30.9 lbf
C. Elliptical shoulders	114 cm <sup>2</sup>		111.1 N	25.0 lbf	166.7 N	37.5 lbf

When making these calculations, some basic relationships to remember are:

$$A_{\text{circle}} = \pi r^2$$

$$A_{\text{ellipse}} = \pi r_1 \times r_2$$

$$1 \text{ mm Hg} = 133.3 \text{ Pa (or N/m}^2\text{)}$$

$$1 \text{ N} = 0.2248 \text{ lbf}$$

Therefore, the entire calculation (with appropriate units) becomes:

$$F (\text{N}) = P (\text{mm Hg}) \times (133.3 \text{ Pa/mm Hg}) \times$$

$$A_{\text{model}} (\text{m}^2)$$

Area in m<sup>2</sup> is found by dividing the area in cm<sup>2</sup> by (100)<sup>2</sup>.

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As a fetus proceeds through the cardinal movements of labor, it will enter the maternal pelvis through the pelvic inlet, pass through the bony structures of the pelvic girdle, and then move through the pelvic outlet before crossing the perineum. Its shoulders must pass at least two bony structures that can impede forward motion—the sacral promontory in the posterior aspect of the birth canal and the symphysis pubis on the anterior side. Impact with these structures may be transient in nature—and cleared through the action of maternal forces alone—or may require maneuvers to alleviate the impaction. Contact between the posterior shoulder and the sacral promontory occurs before the head delivers; the head will typically be at the level of the mid-pelvis (9). It is not possible to diagnose or evaluate such an impaction, and the clinician must proceed with a delivery plan on the basis of observable indicators of progress.

Contact between the anterior shoulder and the symphysis pubis typically occurs as the head delivers over the perineum. As the anterior shoulder impacts the symphysis, the head and neck will advance as a result of the next contraction and may retract back on the perineum. This phenomenon of head-neck retraction tightly against the perineum is commonly referred to by clinicians as a “turtle sign.” From such signs, shoulder dystocia may be suspected. Conversely, no turtle sign may be observed (possibly due in part to variations in maternal anatomy that mask the retraction), and the shoulder may be cleared from the impaction with the next push or contraction solely as a result of maternal (endogenous) forces.

In an uncomplicated vertex delivery, all applied forces will move the infant as a single structure with minimal differential motion between the regions of the body. However, when one of the shoulders is restrained by the bony pelvis, any forces that continue to advance the head and neck will cause a stretch in the brachial plexus. Forces applied through the fetal pelvis by maternal contractions and Valsalva maneuvers will be transmitted up the fetus’ spine. The spine

is a column that transfers force along its length—in this case, from the sacrum to the cervical region and the head. An impaction of one of the shoulders will not eliminate the force transmitted from the pelvis to the spine. Therefore, if a shoulder is restrained, maternal forces will continue to move the head and neck forward, widening the angle between the neck and shoulder and causing traction on the brachial plexus (1).

### Evidence of the Effect of Endogenous Forces

In 1992, Jennett’s retrospective analysis challenged the premise that all incidences of NBPP are due to excessive lateral traction applied by the clinician (10), later following up with a detailed review of four cases in which evidence pointed to causes other than lateral traction as the reason for persistent NBPP (11). Both malpositioning in utero, either long-term or during delivery, and stretch of the brachial plexus when the posterior shoulder impacted pelvic structures were identified as potential causes of the nerve damage. In 1997, Ouzounian and colleagues described eight cases of persistent NBPP that occurred without clinically ascertainable shoulder dystocia on the affected side (12). Four infants were delivered without any indication of shoulder dystocia; four others sustained a posterior arm injury when anterior shoulder dystocia was documented. They pointed to these cases as evidence of a mechanism of injury other than clinician-applied traction.

In 1999, Gherman and colleagues reviewed the clinical literature published since 1984 on NBPP. They found that infants with NBPP without clinical evidence of shoulder dystocia seemed to be of lower birth weight, had more persistent injuries, were more likely to have been delivered following a short second stage of labor (perhaps indicating insufficient time for the fetal shoulders to rotate to a more favorable position), and more frequently had posterior shoulder involvement (13). Recognizing that a portion of incidences of NBPP occurring in the absence of a recognized shoulder dystocia may represent a lack of diagnosis or incomplete documentation of the event,

Gherman and colleagues hypothesized that several mechanisms of injury may occur, depending on the characteristics of the fetus and the delivery, for example:

- Continued movement of the head following impaction of the anterior shoulder behind the symphysis pubis or impaction of the posterior shoulder on the sacral promontory
- Normal downward traction applied by the physician in the presence of observed shoulder dystocia
- Compression of the brachial plexus against the symphysis pubis
- Abnormal intrauterine pressure arising from uterine anomalies or uterine hypertonicity

During a posterior shoulder impaction at the level of the sacral promontory, it is not possible for the clinician to apply extraction forces that are often put forth as the cause of the injury because the head has not delivered. The high rate of posterior shoulder involvement in deliveries that do not involve shoulder dystocia indicates that severe and persistent injuries may occur to the brachial plexus without the clinician's application of traction during the delivery (13). In this regard, "nondystocia" or posterior arm cases have often involved assertions that shoulder dystocia occurred, but was undocumented. An alternative explanation is that the fetus may have encountered an obstruction within the maternal pelvis during descent through the birth canal but cleared that obstruction with the next contraction or maternal push. This explanation eliminates the clinical diagnosis of shoulder dystocia because there was no difficulty in delivering the shoulders subsequent to the emergence of the fetal head. This possibility was supported by Towner in her study of instrumented deliveries and neonatal injury, wherein she describes brachial plexus stretch as a phenomenon with "shoulder dystocia at the pelvic inlet" (14).

In further support of this concept, Allen and Gurewitsch published a case report that involved an infant who sustained transient, posterior NBPP with no application of traction (5). The intrapartum management was substantiated by an accompanying video documenting that no traction was applied by the birth attendant during the delivery process. These same authors later published an article specifically analyzing the differences in NBPP related to clinically evident shoulder dystocia and deliveries without shoulder dystocia (4). They found that 90% of their persistent injury cases were related to documented shoulder dystocia—suggesting that 10% of permanent injuries either had no documentation of or no occurrence of associated shoulder dystocia.

No published clinical or experimental data exist to support the contention that the presence of persistent (as compared to transient) NBPP implies the application of excessive force by the birth attendant. A single case report describes a case of persistent NBPP in a delivery in which no traction was applied by the delivering physician and no delay occurred in delivering the shoulders (15). Therefore, there is insufficient scientific evidence to support a clear division between the causative factors of transient NBPP versus persistent NBPP.

A bioengineered laboratory model was created to examine the mechanical fetal response to the cardinal movements of labor generated with endogenous maternal forces (2). An operator-controlled application of force was used to propel the fetus through the pelvis. The model was designed to measure stretch in both the anterior and posterior brachial plexi. The study compared neck extension, head rotation, and brachial plexus stretch in three types of deliveries: routine, unilateral shoulder dystocia (anterior), and bilateral shoulder dystocia (anterior and posterior) (2). Greater posterior brachial plexus stretch was evident in routine deliveries than in either type of shoulder dystocia delivery. Increasing the bisacromial diameter (from 11.9 cm for routine deliveries to 12.9 cm in bilateral shoulder dystocias) to mimic obstruction was a potentially confounding factor when comparing the results

among and between the three types of deliveries. This change in shoulder properties is likely to have affected the stiffness of the shoulder, which is important in determining both the force necessary to deliver the fetus and the response of the brachial plexus. Still, these data add important information about the potential for endogenous forces to modify baseline brachial plexus stretch before any application of exogenous forces by the clinician.

## Exogenous Forces

In vaginal deliveries, exogenous forces (those applied directly to the fetus by a clinician's hands or through an instrument) are used to assist the natural, maternal forces to effect delivery. In an instrumented delivery, forces are applied while the entire fetus is still within the birth canal. Once the presenting part delivers over the perineum, endogenous force may be sufficient to achieve delivery; if not, the clinician can also apply a guiding or traction force directly to the fetus (to the head in a vertex presentation).

Exogenous forces are often separated into two categories: 1) axial traction and 2) lateral bending. As previously noted, axial traction refers to a force applied in line with the fetal cervicothoracic spine (Fig. 3-1; see color plate) (16). Because the position of the infant within the maternal pelvis will be at some angle relative to the horizontal plane (eg, the delivery table) during the final cardinal movements of labor, axial traction is generally applied in a direction or vector below the horizontal plane (also referred to as downward axial traction as distinguished from downward lateral traction or lateral bending). The key principle to minimize stretching of the brachial plexus through the application of this exogenous force is to avoid lateral bending of the neck—that is, moving the fetus' head out of alignment with the fetal cervicothoracic spine, whether toward the floor or the ceiling (1).

To understand the effect of exogenous forces on a fetus during delivery, it would be helpful to quantify the amount of force applied by clinicians in various delivery situations. Multiple

attempts have been made over the past 20 years to determine this force. In 1991, Allen and Gonik described the exogenous forces applied by a single delivering physician in 29 deliveries (17). They placed fingertip sensors under sterile gloves on the right hand of the physician to measure the contact force between the hand and the fetus' head. This contact force was then correlated with a traction force through a laboratory system. The delivering physician was asked to characterize subjectively each delivery as normal, difficult (but not involving shoulder dystocia), or involving shoulder dystocia. The measured peak delivery force for a difficult delivery (mean of 69 N [15.5 lbf]) was significantly higher than for a normal delivery (mean of 47 N [10.6 lbf]). Two shoulder dystocia cases were encountered, with a mean peak delivery force of 100 N (22.5 lbf). The infant weights in the shoulder dystocia cases were almost identical; one infant had transient NBPP, and the other was uninjured. This study provided new insight into physician-applied forces associated with fetal extraction. However, the accuracy of the measured forces was not validated. Because this study measured contact forces and not actual axial or bending forces, there will be a discrepancy in the calibration.

Poggi and colleagues measured *in situ* clinician extraction delivery force in 27 deliveries in which the woman was randomly assigned to lithotomy or McRoberts positioning (18). The force measurement system was similar to that used in the Allen and Gonik study (17). Two cases of shoulder dystocia that occurred during these deliveries were resolved by the clinicians using forces that were not statistically different from the deliveries without shoulder dystocia. They found that the average peak force measured was 32 N (7.2 lbf) for the lithotomy position group and 35.6 N (8.0 lbf) for the McRoberts position group. A further study by Poggi and colleagues revealed that the applied force needed to assist delivery was greater in women who had received epidural anesthesia than those who had not (34.3 N [7.7 lbf] versus 17.3 N [3.9 lbf]) (19). Again, in this study, no difference in peak delivery force

was identified for the two deliveries in which mild shoulder dystocia occurred. Thus, the three published studies in which delivery force has been directly measured in a clinical setting (admittedly involving only two delivering clinicians) showed a range between 17.3 N (3.9 lbf [plus or minus 2.0]) and 99.6 N (22.4 lbf [plus or minus 0]) and a standard deviation for each experimental group of less than 8 N (2 lbf). These studies provide a limited assessment of exogenous forces but do not address the angle at which forces were applied.

In 1994, Allen published the results of a laboratory study that was designed to analyze the forces applied by clinicians in routine, difficult, and shoulder dystocia deliveries in an attempt to determine how an average obstetrician might characterize a delivery based on their perceptions of applied force (6). A physical model was constructed that allowed for direct measurement of both vertical (downward) force and horizontal (outward) force applied by the birth attendant, as well as the bending moment (force resulting in bending of the neck). The model did not allow for axial or rotational movement or delivery of the fetus. Thirty-nine clinicians were asked to apply the forces that they perceived they would apply in three types of deliveries. The investigators concluded that clinicians tend to apply a greater amount of force along the axis of the fetus' spine as they perceive the delivery to be more difficult. The vertical component of the force also increased (indicative of traction not aligned with the fetus' spine), but to a lesser extent, as did the length of time during which force was applied. Bending, however, was seen to decrease between routine and more difficult deliveries. Again, although new insights can be gleaned from this study, conclusions directly applicable to the clinical arena are limited. With no feedback on whether the applied force was sufficient to deliver the fetus, it is difficult for the birth attendant to determine how the simulation corresponds with the force they would apply clinically. In addition, a single, nonrandomized attempt at each delivery type did

not allow the clinicians to adjust to the response of the system in making their self-assessments.

Crofts and colleagues used a high-fidelity training mannequin in hospitals in the United Kingdom to review shoulder dystocia delivery techniques (20, 21). The system was used to evaluate clinicians both before and after specific training in managing shoulder dystocia. The measured forces ranged from 7 N to 169 N (1.6–38 lbf) in one study (20) and 36 N to 254 N (8.1–57.1 lbf) in another (21). These same investigators later published a study of 113 simulations in which shoulder dystocia was encountered and could only be relieved by delivery of the posterior arm (22). Two thirds of the participants applied a force greater than 100 N (22 lbf) at some point in the delivery scenario (range: 6–250 N [1.3–56.2 lbf]). In these simulations, maximum force application occurred in the last half of each delivery process in most cases. The majority of the clinicians (72%) applied less than 150 N (33.7 lbf) of force at all points during the delivery, and the mean diagnostic force (the traction applied initially to identify the presence of shoulder dystocia) was found to be only 47 N (10.6 lbf). Although these studies do not directly match the clinical environment, they do give insight into clinician behavior at delivery.

## Evidence for the Effect of Exogenous Forces

### Observational and Epidemiologic Studies

Because of the difficulty in conducting biomechanical experiments within the delivery suite, observational and epidemiologic studies have been used to provide some important, although limited, insights into the pathophysiology of NBPP. It is reasonable to assume that most of these cases result from forces experienced during the birthing process. In some instances, however, the injury may be unrelated to the delivery, resulting from congenital abnormalities, fetal malpositioning within the uterus, uterine abnormalities, or postpartum events. These less common causes can result in damage to the nerve resulting from intrauterine arm malpositioning, humeral osteomyelitis

resulting in ischemia to the nerve, in utero viral infections, hemangioma, exostosis of the first rib, neck compression, or neoplasm (tumor) formation (23).

In his original description of an upper plexus palsy as it occurs in infants, Erb indicated that tension on the nerve during delivery resulted in the nerve injury—in particular during the Prague maneuver, designed to deliver a child in the breech position (24). Many of the initial postulates regarding the cause of birth-related NBPP are based on case studies and epidemiologic trends. For example, Buschor and colleagues found that the rate of permanent injuries was significantly higher in vertex deliveries (60%) than in breech presentations (9%) (25). On the basis of this finding, they hypothesized that the injuries in breech deliveries were more compressive in nature, resulting from pressure against the nerve by the clavicle, whereas NBPP in a vertex presentation was caused by traction. However, Rubin found that residual injury at 3 weeks was present in 50% of breech deliveries, compared with only 22% of cephalic deliveries (26). This contradiction in the data illustrates the difficulty in using epidemiology in isolation as a means to understand the mechanism of injury.

For most of the 20th century, it was routinely accepted that NBPP was caused by clinician-applied traction during the delivery process, despite an absence of clinical data supporting this contention. This premise remained uncontested because increased traction, despite the potential for brachial plexus injury, was deemed preferable to asphyxia resulting in a brain-damaged neonate or dead fetus. Clinical textbooks recommended against applying significant traction but did not include data to define the amount of force.

Toward the end of the 20th century, clinical data began to appear indicating that mechanisms other than clinician-applied traction may play a role in the genesis of NBPP. In 1981, Hardy published a 9-year review from New Zealand of infants with NBPP (27). Of 36 cases, only 10 were the result of documented shoulder dystocia. A 1986 study published by McFarland and colleagues

found that low Apgar scores occurred significantly more often in infants with NBPP compared with a control group (28). Low Apgar scores potentially indicate fetal depression, which can result in both reduced muscle tone and lower resistance to any applied forces. Precipitous second stage of labor increases the likelihood that normal truncal rotation will not occur and the fetal shoulders will enter the pelvic inlet in a nonoblique plane. In 1988, Acker and colleagues first described a precipitous second stage of labor as an independent risk factor for NBPP (29). This finding was later confirmed by Poggi and colleagues, who found that 21 of 74 (28%) infants with permanent NBPP had a precipitous second stage of labor (30). Together, these data describe other variables, in addition to force, that might influence susceptibility to NBPP.

Interventions intended to reduce delivery forces have not been shown to affect the rate of NBPP, which further suggests that other factors are involved. Gherman and colleagues reviewed the literature in 2006 to examine trends relating to the management of shoulder dystocia and the occurrence of NBPP (31). They found that the rate of NBPP remained reasonably constant between the early 1980s and the 1990s, despite the later widespread use of the McRoberts maneuver and the abandonment of fundal pressure to reduce shoulder dystocia. The fact that the rate of NBPP has remained nearly constant despite significant changes in clinical practice indicates that it is not simply clinician-applied traction that is responsible for these injuries.

In Sweden, Mollberg and colleagues implemented a 24-question survey for each of more than 31,000 deliveries from 1999 through 2001 (32, 33). Among the questions posed, clinicians were asked to mark the level of force they applied on a semiquantitative scale. Within the 31,000 deliveries, 18 incidences of persistent NBPP and 80 incidences of transient NBPP (defined as recovery by age 18 months) occurred (33). The authors concluded that the persistent injuries were associated with a perceived higher level of downward traction on the head than the transient injuries.

In addition, the transient injuries were associated with a perceived higher level of downward traction than used for a control set of uninjured infants. It should be noted that 94% (17/18) of the cases of persistent NBPP were associated with the application of fundal pressure after the head delivered, compared with 8% of deliveries for the infants whose injury spontaneously recovered and 6% of the control deliveries. Despite the conclusion by Mollberg and colleagues that forceful downward traction was causative, it is not possible to separate out the effect of increased traction from the effect of increased expulsive force induced by the application of fundal pressure.

### Cadaveric Studies

As prospective studies of NBPP are neither ethical nor feasible, studies on cadavers can provide a potential surrogate through which to investigate the mechanics of NBPP. Although the results of such investigations are useful in raising clinical questions, the relationship between the *in vivo* occurrence of NBPP and *in vitro* experiments on cadavers is speculative. In 1916, Sever reported on NBPP to the orthopedic community (34). At that time, 451 cases of NBPP had been reported in the literature between 1872 and 1916. Sever briefly described his own cadaveric experiments, in which the brachial plexus was exposed and forces applied to the head with the arm in various positions. He noted that “considerable force” was required to disrupt spinal nerves C-5 and C-6 but did not provide any quantitative data. He was unable to injure C-8 and T-1 unless the arm was abducted away from the body. He described the injury to C-5 and C-6 as occurring first by partial tearing or rupture of the neural sheath followed by fraying of the nerve just inside of the sheath.

In 1922, Adson described a cadaveric study on adult brachial plexus structures (35). This key work described the viscoelastic nature of the brachial plexus; forces that could easily be sustained at low rates of application could tear the nerve if applied quickly. However, the level of forces that Adson reported for visible tearing of the nerve are likely to be much greater than necessary to cause functional damage.

In 1954, Morris gave an oral presentation in which he made some observations that remain pertinent to today’s theory on the mechanisms of injury to the infant brachial plexus (36):

- Traction applied while protecting the axis of the spine is less likely to injure the plexus than when bending of the neck occurs.
- Lateral bending significantly increases the tension in the nerve when traction is applied.
- Rotation of the neck in addition to lateral flexion further increases the tension on the nerve and the risk of injury.
- Jerking is more dangerous than smoothly and gradually applied traction.

In 1979, Metaizeau and colleagues tested nine infant cadavers in a study designed to assess progression of brachial plexus injuries as a result of bending the infant’s neck (37). These cadavers were placed in a position to simulate shoulder dystocia, the brachial plexus was visualized, and forces were applied using a pulley system. The authors reported that the first “objective lesion” occurred between 20 kg and 40 kg of applied mass. Although the study does not provide solid biomechanical injury data for the neonatal brachial plexus, it did document the progression in the injury that occurred. As lateral traction was applied, the upper plexus was the first portion of the complex to be damaged—generally resulting in nerve root rupture. With continued lateral loading following C-5–C-6 rupture, avulsion of the C-7 and C-8 roots occurred.

The cadaveric work to date to examine the *in situ* response of the brachial plexus has been quite crude by today’s standards of biomechanics. The studies do lend insight into one of the injury mechanisms of the brachial plexus—the application of lateral bending. However, they do not provide a complete picture of how and why NBPP may occur during delivery. In fact, examining only one direction of force application (lateral bending) without paying equal attention to

other potential causes of brachial plexus stretch provides only a partial picture. It is inappropriate to conclude that lateral bending is the only cause of the injury on the basis of these early studies when similar research has not examined other mechanisms.

#### **Studies Using Physical and Computer Models**

Because of the limitations of clinical and cadaveric studies, engineering models have been used to study the forces of labor and their effects on brachial plexus injury. Physical models provide a “hands-on” system that clinicians and researchers can use. Computer models build a virtual representation of the birth process and the fetus and follow accepted physical laws. The goal is to develop a model that mimics the anatomy, biomechanics, and physiology of the actual system as closely as possible. The challenge with a physical model (compared with a computer model) is finding materials with the same or at least similar properties as the tissues of interest. In a computer model, these properties can be defined based on known values and mathematic relationships.

To better understand the relationship of exogenously applied force and its effects on the fetus, a physical laboratory model of childbirth was developed (38). Physician-applied forces were measured using glove sensors that had been calibrated separately to assess traction. The model demonstrated that as the dimensions of the fetus were made larger, extraction forces to deliver the anterior shoulder increased. Stretch in the brachial plexus tended to increase at a slightly higher rate than the neck extension as the applied extraction force increased. Brachial plexus stretch of between approximately 10 mm and 25 mm was predicted for applied traction between 40 N and 120 N (9.0–26.9 lbf) (38); however, the model did not allow for calculation of strain, nor was an assessment of its biofidelity possible (ie, how lifelike or realistic the model was). In an updated version of this model, a new group of investigators examined the difference in clinician-applied forces and brachial plexus stretch when either the McRoberts position or the Rubin maneuver (oblique position-

ing) was applied in a shoulder dystocia delivery (39). Despite bioengineering limitations that did not allow for direct clinical applicability, the model was useful to examine trends in fetal response to exogenously applied extraction forces. Significantly less traction was required to deliver the model infant following the Rubin rotation than in the McRoberts position. In addition, the extension in the brachial plexus element was reduced significantly using the rotational maneuver.

#### **Comparing Effects of Endogenous and Exogenous Forces**

A sophisticated three-dimensional computer model (MADYMO) was developed to investigate both endogenous and exogenous delivery forces in one system and their effects on the fetus (16). The maternal pelvis model corresponded with the measurements of a 50th percentile gynecoid pelvis, and the fetal model matched the size of a 90th percentile newborn. The level of endogenous or exogenous force applied to achieve delivery could be increased in a step-wise fashion. For this model, the predicted forces required to achieve delivery were 400 N for maternally generated (endogenous) forces and 100 N for clinician-applied (exogenous) forces. The study found that contact force at the base of the fetal neck against the maternal symphysis pubis was more than two times higher because of maternal endogenous forces when compared with exogenous forces. Of interest, when McRoberts positioning was applied to the computer-generated maternal pelvis, the exogenous force required to achieve delivery was reduced by 50%, consequently also reducing the resultant contact force at the fetal neck overlying the brachial plexus. With lateral bending of the fetal neck during exogenous extraction efforts (rather than axial traction), 30% more force was required to achieve delivery of the shoulders.

The same investigators further developed the MADYMO computer model to look more specifically at brachial plexus response (1). The maternal pelvic model was unchanged, but the fetal

model was modified on the basis of animal models to include shoulder and neck structures that more closely represented a human fetus (40, 41). As in the previous model, a greater endogenous force was required to deliver the fetus than if an exogenous force was applied. The force necessary to achieve delivery was reduced compared with the first version of the model (16), which was the result of the more biofidelic and deformable shoulder in the new model. The new maternal delivery forces (125 N [28.1 lbf]) are well within the range anticipated based on measurements of intrauterine pressure (42).

In the updated computer model, the predicted brachial plexus stretch was greatest for clinician-applied forces when the fetus' neck was allowed to bend downwards (18.2%) (1). If the spine's axis was protected during applied traction (axial traction), the stretch was reduced to 14% in the lithotomy position, and reduced even further in the McRoberts position to as low as 6.5%. In contrast, the stretch that occurred as a result of maternal endogenous forces as shoulder dystocia occurred was predicted to be 15.7% in the lithotomy position for a force that resulted in delivery, dropping slightly with McRoberts position. In the case of a maternal endogenous force that was not sufficient to deliver the model fetus (100 N [22.5 lbf]), the predicted stretch in the brachial plexus was even higher than if delivery occurred (18.2%). These findings indicate that shoulder dystocia may, in and of itself, induce a similar amount of stretch in the brachial plexus as lateral bending of the fetus' neck.

The MADYMO model has most recently been used to evaluate the benefit of other clinician maneuvers on reducing the stretch in the brachial plexus during delivery (43). Suprapubic pressure, oblique positioning, and delivery of the posterior arm all reduced both the clinician-applied force required to deliver the fetus and the resulting stretch in the brachial plexus. In the MADYMO model, delivery of the posterior arm resulted in the greatest decrease in required traction and resulting stretch,

with a required clinician-applied delivery force of only 30 N (6.7 lbf) and a predicted stretch to the brachial plexus of 5.3%.

## Biomechanics of Nerve Injury

Nerve injury may involve both traction and compression pathomechanic events. For example, compression on a nerve will make it more susceptible to injury because the compression may damage the epineurium (the outer sheath of the nerve structure) and the axonal tissue. Once damaged, the nerve has the potential to fail at a lower amount of applied traction. In addition, a compressive force to the shoulder can functionally shorten the nerve segment by pinning it in place or can induce traction to the brachial plexus by displacement at the middle of the nerve span because the ends of the string-like nerve are fixed both proximally and distally. Compression alone can result in permanent injury if the compression is maintained for a sufficient period and is of a significant magnitude (44, 45).

Because stretch of a nerve is a recognized mechanism of brachial plexus rupture or avulsion during the birth process, it is important to explore what can cause sufficient stretch of the nerve to lead to this type of injury. From a biomechanical perspective, a necessary condition for significant stretch to the brachial plexus is the widening of the angle between the fetus' head and neck. If the fetus' shoulder comes in contact with an obstruction such that the shoulder is immobilized, while the fetus' head and spinal column continue to move, the angle between the fetus' head and neck will be widened. The obstruction may result in either shoulder dystocia (resulting in delay or difficulty in delivering the anterior shoulder) or temporary impact by either the anterior or posterior shoulder on the symphysis pubis, the sacral promontory, or other pelvic structures. There is some evidence that the cardinal movements of labor alone may cause stretch in the brachial plexus (2), but the extent of this stretch requires more investigation. Thus, the clinical and biomedical engineering evi-

dence supports the assertion that when a shoulder is restrained either transiently or during a more significant impaction, both maternal forces and clinician forces, if applied, will stretch the brachial plexus.

## Injury Threshold

With regard to extraction or expulsion forces, one group of investigators has suggested that the threshold for NBPP in the human is 100 N (22.5 lbf), on the basis of a single case of transient NBPP (17) and limited laboratory modeling data (38). Significant variation exists between individuals, both in terms of mechanical properties and anatomy. Because of the nonlinear behavior of tissues such as nerve tissue, an estimate of the force needed to cause a nerve rupture cannot be directly established. Despite the injury threshold suggested by mechanical models, physician-applied forces measured with simulators commonly exceed this hypothesized cutoff of 100 N even for routine deliveries (6, 22), and yet the NBPP rate remains low (46, 47).

Additionally, some authors have reviewed studies on delivery forces applied through forceps and have postulated that infants can withstand delivery forces up to 200 N (45 lbf) without NBPP (48). Deliveries without shoulder dystocia were included in these forceps cases, so infants were presumably able to move down the birth canal as a result of the applied force (49, 50). With an obstructed delivery, differential motion can occur between body parts, resulting in selective tissue stretch or compression at or beyond the site of obstruction. Therefore, the fact that 200 N of force could be applied to a fetus to effect delivery in the absence of clinical shoulder dystocia does not establish a permissible or “safe” traction force in the presence of shoulder impaction with the maternal pelvis.

The nerve tissue properties of the newborn brachial plexus have not been adequately studied to establish thresholds for damage based on either applied force or resulting stretch. The use of

data from cadaveric studies, in vitro experiments, and animal models has limitations. Nerve tissue deteriorates quickly postmortem, making cadaveric studies on the biomechanical properties of human nerves problematic. In addition, because healthy human nerves are generally not resected during surgery, such specimens are not readily available for study. Finally, there may be significant differences between adult and newborn nerve tissue that further complicate comparisons.

With these limitations, some general concepts may be gleaned from the existing data. Tubbs and colleagues studied the cranial nerves of six adult cadavers immediately postmortem to determine their tensile load at failure (51). They found a fourfold variation in the range of failure forces for cranial nerves III through XII, with several of the nerves failing at levels not in proportion to their diameter. These results suggest there may be a range of failure responses, with properties depending on something other than simply the cross-sectional area of the nerve. In 1993, Marani and colleagues published preliminary data on the tensile testing of adult brachial plexus nerves (52). The majority of the specimens were fixed in formalin, a preservative known to affect the soft tissue mechanical properties. When a very low rate of deformation was applied to the unfixated specimens, the strain at rupture was approximately 33%. For high rates of deformation, the strain at rupture was reduced to only 3–5%. These findings indicate the viscoelastic nature of nerves, in which applying force at an increased rate (ie, faster), will cause the structure to fail at a lower strain.

Studies have been conducted on peripheral nerves and nerve roots in a variety of animal models. The deformation response is typically nonlinear, showing the standard, initial, low stiffness that is seen in most soft tissues followed by increasing stiffness as deformation increases. In 1992, Kwan and colleagues reported on the functional response of rabbit tibial nerves exposed to tension (53). Slow application of strain demonstrated that 6% strain caused a 70% reduction in

conduction amplitude, with an almost complete recovery following removal of the applied force. With a 12% strain, however, the conduction decreased rapidly and was interrupted completely by 60 minutes of strain and did not recover fully over the next 60 minutes—leaving a 60% deficit. This failure of recovery is presumptive evidence of mechanical damage to the nerve, which may or may not have recovered beyond the study time interval. A similar study by Takai and colleagues on rabbit brachial plexus nerves 10 years later found that nerve conduction was blocked at an equivalent of 8% strain (54). These studies used a very slow application of strain; faster applications of the same amount of strain are likely to result in more injury through mechanical damage.

A subset of adult animal studies has examined the actual physical failure response of peripheral nerves. Using sciatic, tibial, spinal, and brachial plexus nerve types, these studies reported nerve failure at mean strains ranging from 24% to 48% (41, 54–56). There is significant variability among animals within the same species, and portions of the studied population experienced nerve rupture at strains between 11% and 20% (56). Using rabbit tibial nerves, Rydevik and colleagues documented how much strain a nerve undergoes simply based on anatomic placement compared with a nerve under no strain (ie, *in vitro*) (41). The rabbit tibial nerves studied showed 11% *in situ* strain in a resting state. If *in situ* nerves are already stretched to some degree, less additional strain will be required to reach the laboratory-measured failure strains than if the nerve started at its zero-tension length.

## Biomechanics of Neonatal Brachial Plexus Palsy in the Absence of Clinical Shoulder Dystocia

An obstruction of part of the fetal body allows a difference in the motion between the head and shoulders and is required for the brachial plexus to stretch significantly when delivery forces are applied. This obstruction is obvious in the case of recognized shoulder dystocia—the anterior shoul-

der becomes impacted on the symphysis pubis. In cases of NBPP without clinically evident shoulder dystocia, there are four basic scenarios: 1) anterior shoulder impaction, 2) posterior shoulder impaction, 3) breech presentation, and 4) cesarean delivery.

For anterior shoulder NBPP, the affected shoulder must come in contact with a portion of the maternal pelvis. Given the cardinal movements of labor, such a situation will most likely be the result of the shoulders not rotating spontaneously to an oblique orientation, at which time the anterior shoulder contacts the symphysis pubis. However, in some deliveries, it is possible for maternal forces to push the fetus clear from this impaction (1). In this case, there is no delay in delivery of the body following the head, no need for maneuvers, and no diagnosis of shoulder dystocia. The brachial plexus may have already stretched substantially, however, which can lead to either transient or persistent NBPP in an infant with a relatively low injury threshold (1).

In the case of posterior shoulder NBPP, the obstruction is believed to be caused by the shoulder interacting with the sacral promontory (5) or the hollow of the sacrum (11). In the case of the sacral promontory, the head will not yet have delivered when this contact occurs. Therefore, for deliveries in which no instruments are used, only maternal forces will determine when the impaction and resulting stretch in the brachial plexus occurs. In breech deliveries, stretch to the brachial plexus may occur if the head is retained by the maternal pelvis after delivery of the fetus' body. This stretch is likely to result from a combination of the fetus' position and clinician-applied traction to the shoulders used to extract the head.

The cause of obstruction that results in brachial plexus stretch in cesarean delivery is less clear. For some infants, the stretch may be the result of prolonged malpositioning in utero. If the woman labored before cesarean delivery, maternal forces may have affected the fetus through contact of the fetus' shoulders with an obstruction. The obstruction may have resulted from the maternal

bony pelvis or unusual resistance from maternal soft tissues. Another possibility is that during extraction through the hysterotomy site, lateral flexion of the fetal head in combination with existing fetal positioning led to brachial plexus stretch and injury.

When the structure impeding the motion of the fetus' shoulders (or head in the case of breech delivery) comprises soft tissues, the stretch in the brachial plexus will be less than with a bony structure. The applied force will cause deformation in the obstructing soft tissue as well as the connective tissues of the fetus, so that a smaller portion of the force will be transmitted directly to the brachial plexus to cause the nerve to stretch. Therefore, under equivalent endogenous or exogenous forces, soft tissue obstructions are less likely to result in either transient or persistent fetal injury than if contact occurs with the bony pelvis.

In addition to research within the obstetric community, the pediatric, orthopedic, and neurologic literature now stress that the existence of NBPP following birth does not a priori indicate that exogenous forces are the cause of this injury. The pediatric neurologic community also has reviewed the literature on causation and has similarly concluded that, "The obstetrician's efforts to relieve shoulder dystocia are not the whole explanation for brachial plexus birth injuries. Expulsive forces (ie, endogenous forces) generated by the uterus and the abdominal wall ... may be contributory in many cases" (57).

## Summary

Neither high-quality nor consistent data exist to suggest that NBPP can be caused only by a specific amount of applied force beyond that typically used by health care providers and experienced during a delivery without NBPP. Instead, much of the data suggest that the occurrence of NBPP is a complex event, dependent not only on the forces applied at the moment of delivery, but also on the constellation of forces (eg, vector and rate of application) that have been acting on the fetus

during the labor and delivery process, as well as individual fetal tissue characteristics (eg, in situ strain and acid-base balance).

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# Chapter 4

## Neonatal Brachial Plexus Palsy and Shoulder Dystocia

### Shoulder Dystocia: An Overview

A commonly accepted definition of shoulder dystocia is a delivery that requires additional obstetric maneuvers following failure of gentle downward traction on the fetal head to effect delivery of the shoulders (1). This definition by the American College of Obstetricians and Gynecologists is generally consistent with that of the Royal College of Obstetricians and Gynaecologists, although the two organizations differ in some respects on recommended management (Table 4-1; see also Appendix A and Appendix B) (2, 3). Clinically, shoulder dystocia is diagnosed after delivery of the fetal head when the anterior shoulder fails to emerge under the pubic rami as a result of impaction of the fetus' anterior shoulder behind the pubic symphysis. Anterior shoulder neonatal brachial plexus palsy (NBPP) may result from such an impaction; posterior shoulder NBPP is not associated with clinically apparent shoulder dystocia or traction by the birth attendant (3). Retraction of the fetal head against the maternal perineum, accompanied by difficulty in accomplishing external rotation, has been characterized as the "turtle sign."

Although in most cases it is this combination of events that raises suspicion that shoulder impaction against the symphysis pubis has occurred, shoulder dystocia is not formally diagnosed until a trial of downward axial traction has been unsuccessful in delivering the anterior shoulder. One study proposed an objective definition of shoulder dystocia as delivery that involved a head-to-body interval of more than 60 seconds or required ancillary obstetric maneuvers or both, and a prospective evaluation confirmed the utility of this definition (4, 5). In some countries, a "two-step" diagnostic approach (waiting until the next contraction to deliver the shoulders) is recommended by some authors (6, 7). This technique has been prospectively evaluated only with respect to its effect on umbilical artery pH and neonatal outcome (8). However, at this time there is no definition of shoulder dystocia based on this approach.

Recognized shoulder dystocia occurs infrequently, with an incidence ranging from 0.2% to 3.0% of all vaginal deliveries (9). This wide range has been attributed to the inherent subjectivity of the clinician's definition of shoulder dystocia, the extent of reporting, and the differences in the

**Table 4–1.** Differences in the United States' and the United Kingdom's National Guidelines on Shoulder Dystocia ↵

Topic	American College of Obstetricians and Gynecologists (2002)	Royal College of Obstetricians and Gynaecologists (2005)
Shoulder dystocia is complicated by NBPP	4–40%	4–16%
NBPP occurs without shoulder dystocia	34–47%	Substantial minority
Maternal propulsive force as a cause of NBPP	NM	Significant evidence
McRoberts maneuver	Not superior to other maneuvers	Single most effective intervention
All-fours position	NM	An option
Elective cesarean delivery among women without diabetes	Estimated fetal weight greater than 5,000 g	Not recommended at any weight
Rehearsal and skill training	NM	Requirement
Documentation	NM	Recommended
Management of patients with prior shoulder dystocia	Mentioned	NM
Figures of maneuvers	No	Yes
Algorithm for shoulder dystocia management	No	Yes

Abbreviations: NBPP, neonatal brachial plexus palsy; NM, not mentioned.

Chauhan SP, Gherman R, Hendrix NW, Bingham JM, Hayes E. Shoulder dystocia: comparison of the ACOG practice bulletin with another national guideline. *Am J Perinatol* 2010;27:129–36.

study populations. When shoulder dystocia is objectively defined by head-to-body delivery interval (longer than 60 seconds) or the use of ancillary maneuvers or both, the reported incidence approaches 10% (5).

It appears that the incidence of clinically evident shoulder dystocia has been increasing over the past several decades. Mackenzie's study in the United Kingdom from 1991 to 2005 found a

significant linear trend in the incidence of shoulder dystocia, increasing by approximately 3 extra cases per 10,000 births each year (10). Dandolu found that the rate increased steadily with time, from 0.2% in 1979 to 2.1% in 2003. For the state of Maryland, the percentage of deliveries complicated by clinical shoulder dystocia increased from 0.2% (1979–1984) to 0.9% (1989–1994) and then to 2.1% (1999–2003) (11). These

apparent increases may be the result of maternal factors (eg, obesity), fetal factors (eg, macrosomia), or increased reporting. Despite the seemingly increased incidence of shoulder dystocia and increased attention to the clinical management of this event, the incidence of NBPP has remained relatively constant over time.

Multiple studies have clearly documented an increased incidence of shoulder dystocia as neonatal birth weight increases (see [Chapter 2](#)) (9, 12–15). However, the mean birth weight of neonates with recognized shoulder dystocia is not significantly higher than the mean birth weight of all term infants (16). Primary efforts to prevent NBPP, therefore, have been based on attempts to prevent shoulder dystocia itself. Reported management strategies include early induction of labor and prophylactic use of McRoberts maneuver and suprapubic pressure; however, these strategies have not been proved to affect the incidence of NBPP (17–22). The American College of Obstetricians and Gynecologists' Practice Bulletin Number 40, *Shoulder Dystocia*, states that, "Planned cesarean delivery to prevent shoulder dystocia may be considered for suspected fetal macrosomia with estimated fetal weight exceeding 5,000 g in women without diabetes and 4,500 g in women with diabetes" (1).

As evidenced in [Table 4-2](#), transient NBPP has been associated with a range of 1.01% to 16.8% of clinically apparent shoulder dystocia cases. The incidence of persistent NBPP at 1 year of life ranges from 0.54% to 1.6% ([Table 4-2](#)). Maternal and fetal factors associated with shoulder dystocia do not allow for reliable prediction of persistent NBPP. In Poggi's matched, case-control study that assessed maternal weight, body mass index, estimated gestational age, average number of maneuvers, head-to-body delivery interval, operative delivery rate, and prolonged second stage of labor, differences in NBPP were not significant between the two groups (23). Although Gherman and colleagues found that infants with persistent NBPP had a higher mean birth weight (4,519 g versus 4,143 g;  $P < .001$ ) and a greater frequency of birth

weight greater than 4,500 g (38% versus 16%; odds ratio, 0.31; 95% confidence interval [CI], 0.11–0.87), there were no statistically significant differences with respect to multiple antepartum and intrapartum measures (24).

## Management of Shoulder Dystocia

### Communication

Management of shoulder dystocia is a collaborative effort among all members of the delivery team. The delivering clinician is the best person to accurately assess and communicate the details of shoulder dystocia maneuvers. It is important for the delivering clinician to give instructions to the nursing staff regarding the performance of maneuvers such as McRoberts positioning, the application of suprapubic pressure, and the need for a resuscitation team. The woman in labor should be instructed to refrain from pushing during an attempted maneuver. She can then be instructed to resume pushing following performance of a maneuver to allow determination of whether the shoulder dystocia has been successfully relieved.

### Maneuvers

All cases of clinical shoulder dystocia have in common an anatomic disproportion or misalignment between the pelvis of the pregnant woman and the shoulders of the fetus in the specific orientation assumed following delivery of the head (9, 16). However, there is a wide range of normal anatomic variation in the bony and soft tissue structures of pregnant women and fetuses, even within the broad classifications of pelvic shape (eg, gynecoid or android) commonly encountered. Thus, neither the forces applied to the brachial plexus of the fetus as a result of the shoulder dystocia itself (before any maneuvers) nor the forces required to relieve this disproportion or misalignment will be identical in every case.

Notably, when shoulder dystocia is recognized, the fetal neck, shoulders, and underlying structures have, by definition, already been sub-

**Table 4–2.** Neonatal Morbidity Associated With Shoulder Dystocia ↵

Authors	Study period	Number of vaginal deliveries	Number of cases of shoulder dystocia	Clavicle fracture	Transient NBPP	Persistent NBPP after shoulder dystocia
Gherman <sup>1</sup>	1991–1995	50,111	285	9.5%	16.8%	1.4%
McFarland <sup>2</sup>	1986–1994	39,280	276	8.5%	8.5%	NR
Beall <sup>3</sup>	1995–1996	722	99	0%	1.01%	NR
Keller <sup>4</sup>	1983–1989	120	120	1.7%	3.3%	0.83%
Nocon <sup>5</sup>	1986–1990	12,532	185	7.5%	15.1%	0.54%
Sandmire <sup>6</sup>	1985–1994	20,725	188	NR	10.1%	1.6%
Leung <sup>7</sup>	1995–2009	62,295	210	4.4%	8.5%	NR
Mehta <sup>8</sup>	1996–2001	25,995	206	2.9%	9.2%	NR
MacKenzie <sup>9</sup>	1991–2005	79,781	514	NR	8.3%	NR
Hoffman <sup>10</sup>	2002–2008	206,969	2,018	1.9%	3.2%	NR

Abbreviations: NBPP, neonatal brachial plexus palsy; NR, not reported.

<sup>1</sup> Gherman RB, Ouzounian JG, Goodwin TM. Obstetric maneuvers for shoulder dystocia and associated fetal morbidity. *Am J Obstet Gynecol* 1998;178:1126–30.

<sup>2</sup> McFarland MB, Langer O, Piper JM, Berkus MD. Perinatal outcome and the type and number of maneuvers in shoulder dystocia. *Int J Gynaecol Obstet* 1996;55:219–24.

<sup>3</sup> Beall MH, Spong C, McKay J, Ross MG. Objective definition of shoulder dystocia: a prospective evaluation. *Am J Obstet Gynecol* 1998;179:934–7.

<sup>4</sup> Keller JD, Lopez-Zeno JA, Dooley SL, Socol ML. Shoulder dystocia and birth trauma in gestational diabetes: a five-year experience. *Am J Obstet Gynecol* 1991;165:928–30.

<sup>5</sup> Nocon JJ, MacKenzie DK, Thomas LJ, Hansell RS. Shoulder dystocia: an analysis of risks and obstetric maneuvers. *Am J Obstet Gynecol* 1993;168:1732–7; discussion 1737–9.

<sup>6</sup> Sandmire HF, DeMott RK. The Green Bay cesarean section study. IV. The physician factor as a determinant of cesarean birth rates for the large fetus. *Am J Obstet Gynecol* 1996;174:1557–64.

<sup>7</sup> Leung TY, Stuart O, Suen SS, Sahota DS, Lau TK, Lao TT. Comparison of perinatal outcomes of shoulder dystocia alleviated by different type and sequence of manoeuvres: a retrospective review. *BJOG* 2011;118:985–90.

<sup>8</sup> Mehta SH, Blackwell SC, Bujold E, Sokol RJ. What factors are associated with neonatal injury following shoulder dystocia? *J Perinatol* 2006;26:85–8.

<sup>9</sup> MacKenzie IZ, Shah M, Lean K, Dutton S, Newdick H, Tucker DE. Management of shoulder dystocia: trends in incidence and maternal and neonatal morbidity. *Obstet Gynecol* 2007;110:1059–68.

<sup>10</sup> Hoffman MK, Bailit JL, Branch DW, Burkman RT, Van Veldhusen P, Lu L, et al. A comparison of obstetric maneuvers for the acute management of shoulder dystocia. Consortium on Safe Labor. *Obstet Gynecol* 2011;117:1272–8.

jected to abnormal forces preventing the contractions of the uterus from completing the delivery (9, 16). It is not possible to predict which maneuvers will most readily allow completion of the delivery in an individual case. Although no specific sequence of maneuvers has been shown to be superior, a standardized sequence of maneuvers may be valuable within a given institution, as with any medical emergency requiring coordination among multiple health care providers.

When shoulder dystocia occurs, the brachial plexus is already under strain from maternal endogenous forces. Any intervention to effect vaginal delivery (with the exception of episiotomy) involves the application of exogenous forces by the birth attendant that further increases strain on the brachial plexus. The precise physiologic effects of the application of such exogenous forces on the brachial plexus—even if properly performed—are unpredictable (see [Chapter 3](#)).

## New Approaches to Management

In limited studies, the following strategies have demonstrated either a reduction in NBPP or an increased rate of successful resolution of shoulder dystocia. It should be noted that this document does not present comprehensive management strategies. Moreover, no single management strategy is viewed as preferable.

### Prioritization of Posterior Arm Delivery

Delivery of the posterior arm has typically been placed near the end of the obstetrician's clinical algorithm, possibly because of limited training during residency, fear of humeral fracture, or concern that posterior arm delivery represents a more invasive type of maneuver. Recent studies, however, have recommended prioritization of posterior arm delivery following McRoberts position and suprapubic pressure (25, 26). Hoffman reported that this combination of maneuvers was associated with the highest rate of delivery, 84.4%, compared with 24.3–72% for other maneuvers ( $P < .005$  to  $P < .001$ ). After implementation of standardized

training for delivery, Inglis found a significant decrease in the use of McRoberts maneuver ( $P < .01$ ) and an increase in the use of posterior arm delivery ( $P = .02$ ) (25). Leung's review found a success rate of 63.6% for the posterior arm among their initial failed cases. In Grimm's computer modeling study, the greatest effect was seen with delivery of the posterior arm, which showed a 71% decrease in anterior nerve stretch and an 80% decrease in delivery force (26).

### Team Training

When shoulder dystocia is recognized, as in any medical emergency, it is important that communication among all health care providers be clear, succinct, and focused on the essential issues facing the delivery team. These issues include delivery of the infant, avoidance of hypoxic-ischemic central nervous system injury, and minimizing, if possible, additional strain on the brachial plexus beyond that generated by endogenous forces. Process standardization and the use of checklists, teamwork training, crew resource management, and evidence-based medicine have been shown to improve outcomes and quality of care (27).

The study by Inglis and colleagues described a standardized training protocol that covered risk factors, early recognition, management, and documentation (25). Individual hands-on, simulated shoulder dystocia training scenarios were mandatory. After implementing training for maternity staff, the overall incidence of NBPP decreased from 0.4% before training to 0.14% after training ( $P < .01$ ). The rate of NBPP after recognized shoulder dystocia decreased from 30% to 10.67% ( $P < .01$ ) (25). Grobman and colleagues likewise found that introduction of a shoulder dystocia protocol focused on team response was associated with significant improvements in documentation quality and a reduced frequency of NBPP associated with shoulder dystocia. Key components of Grobman's protocol included mandatory didactic sessions for all health care providers on the labor and delivery unit, repeated simulations, and debriefing (28). Simulation and team training are

increasingly being used; ongoing research is evaluating their general application, outcome measures, and frequency of application.

## Simulation

Traditionally, training of health care providers has been based on experiences with actual patients in the clinical setting. It has been suggested that such a training approach may not be ideal for uncommon clinical events, given the lack of opportunity for repetitive experience or for emergent situations, in which the need for rapid response does not lend itself to optimal training.

Simulation provides an opportunity for health care workers to prepare and train for interventions in the event of an emergency (29). Although one of the benefits of simulation is the enhancement of individual performance, an additional benefit is improved teamwork, communication, and mental awareness of the health care team as a whole. Simulation in obstetrics may involve either low-fidelity or high-fidelity simulation. In low-fidelity simulation, participants take part in an event without a mannequin or other technical support that approximates biologic processes during a given medical situation. High-fidelity simulation includes equipment such as a mannequin that mimics events such as shoulder dystocia, blood loss, or hemodynamic instability.

Several investigators have attempted to evaluate whether simulation can reduce the risk of NBPP associated with shoulder dystocia. Many of these investigations have shown that simulation can improve outcomes during subsequent simulations but did not evaluate actual health outcomes (30, 31). There is some evidence, albeit limited, that shoulder dystocia simulation may be associated with fewer cases of transient NBPP. In one observational study in the United Kingdom, Draycott and colleagues studied outcomes associated with the introduction of a mandatory clinical shoulder dystocia simulation for all personnel on a labor and delivery unit. The frequency of transient NBPP associated with actual shoulder dystocia

was significantly lower after health care providers underwent this training (relative risk [RR], 0.31; 95% CI, 0.13–0.72) (32). However, there was no significant difference in NBPP that persisted at 6 months (RR, 0.28; 95% CI, 0.07–1.1) or at 12 months (RR, 0.41; 95% CI, 0.1–1.77). In contrast, MacKenzie and colleagues, in another study from the United Kingdom, were unable to demonstrate a decrease in even transient NBPP after introduction of a training program. Although they found that management of shoulder dystocia improved after training (ie, increased use of the McRoberts maneuver), the frequency of NBPP actually increased by 0.048 per 1,000 vaginal births per year during the period of study ( $P < .01$ ) (10).

The results of two investigations of transient NBPP in the United States have been more consistent with the results of Draycott and colleagues. The study by Inglis and colleagues on team training noted earlier also included simulation (25). Grobman and colleagues demonstrated a reduction in NBPP diagnosed at delivery (from 10.1% to 2.6%;  $P = .03$ ) and at neonatal discharge (from 7.6% to 1.3%;  $P = .04$ ) after introduction of a shoulder dystocia protocol at their institution (28). The introduction of the protocol also involved simulation training, although that training was a low-fidelity type that involved only simulation of the team response, with an actress portraying a patient, and no repetition of maneuvers or delivery using a model pelvis. Although both studies demonstrated a reduction in transient NBPP, neither assessed persistence of NBPP.

Thus, limited data demonstrate that a simulation program may be associated with the reduction of NBPP in the context of clinically apparent shoulder dystocia, but these data are prone to biases inherent in the study design. Regardless of the validity of the results for transient NBPP and the improvement in health care provider competency, there remains no evidence that introduction of simulation can reduce the frequency of persistent NBPP.

## Documentation

Contemporaneous documentation of the management of shoulder dystocia is recommended to record significant facts, findings, and observations about the shoulder dystocia event and its sequelae. Some clinicians use a detailed written or dictated report that includes a description of the maneuvers used, the sequence of maneuvers, and exogenous forces applied. Others use a checklist-type report that contains the critical information needed for documentation, such as the American College of Obstetricians and Gynecologists' Patient Safety Checklist, *Documenting Shoulder Dystocia* (see Appendix C).

## Head-To-Body Delivery Interval

During shoulder dystocia deliveries, shoulder impaction and a prolonged head-to-body delivery interval can lead to birth asphyxia and subsequent neonatal hypoxic-ischemic encephalopathy. In 1998, a case-control study of 15 infants with permanent brain injury after recognized shoulder dystocia demonstrated that a head-to-body interval of 7 minutes or more had 67% sensitivity in predicting permanent brain damage (33). Since then, two larger studies have shown that permanent neurologic injury can occur as soon as 2 minutes after shoulder impaction, although the risk of acidosis or severe hypoxic-ischemic encephalopathy remains low with less than 5 minutes of impaction (34, 35). A prolonged head-to-body interval has also been associated with lower 5-minute Apgar scores (36). A pragmatic approach in all deliveries involving shoulder dystocia would be to minimize delay in delivery as much as possible by implementing appropriate maneuvers once shoulder dystocia is recognized. Cord blood gases may assist in determining the degree of hypoxia and acidemia experienced by the fetus.

Although uncommon, untreated shoulder dystocia can lead to fetal or neonatal death in some cases. The largest study to date on this topic reported an incidence of fatal shoulder dystocia of 0.04 per 1,000 deliveries. In that study, the mean

birth weight was 4,324 g, and 25% of the cases had a birth weight less than 4,000 g. Furthermore, the median time between delivery of the head and the rest of the body in the catastrophic cases was only 5 minutes (37).

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# Chapter 5

## Anatomy and Clinical Presentation of Neonatal Brachial Plexus Palsy

### Brachial Plexus Anatomy

#### Schematic Anatomy

The brachial plexus is a very complex structure that connects the spinal nerves to their terminal branches in the upper extremity. The standard schematic diagram that describes the brachial plexus uses five zones: 1) spinal nerve roots, 2) trunks, 3) divisions, 4) cords, and 5) terminal branches. The complete description of the individual nerves and the muscles innervated by these nerves is beyond the scope of this document and is described in other texts (1, 2). However, understanding the schematic anatomy of the nerves and the muscles they innervate provides a critical foundation from which to understand the clinical presentation of neonatal brachial plexus palsy (NBPP).

The C-5 to T-1 spinal nerve roots contribute to the brachial plexus (Fig. 5-1; see color plate). The C-5 and C-6 roots join to form the upper trunk, the C-7 root forms the middle trunk, and the C-8 and T-1 roots join to form the lower trunk. Each trunk divides into an anterior and posterior division. All three posterior divisions coalesce to form the posterior cord. The anterior divisions from the upper and middle trunk form the lateral cord, and

the anterior division from the lower trunk forms the medial cord. The posterior cord ultimately branches into the terminal branches of the axillary and radial nerves. The lateral cord and medial cord each contribute to the median nerve. In addition to its contribution to the median nerve, the lateral cord terminates in the musculocutaneous nerve, and the medial cord terminates in the ulnar nerve.

A number of terminal branches (nerves) arise from various zones of the basic structure; knowing these branches and their innervated muscles allows localization of a lesion (Table 5-1) (2). For example, the dorsal scapular nerve arises proximally from C-5, and the long thoracic nerve arises from the nerve roots of C-5 to C-7. Lack of function of either nerve implies a proximal injury of the brachial plexus at the level of the nerve roots. Similarly, the phrenic nerve arises from C-3, C-4, and C-5 such that concurrent diaphragmatic paralysis also is consistent with a proximal lesion of the brachial plexus. The upper trunk gives origin to the suprascapular nerve; lack of supraspinatus and infraspinatus function in the context of deltoid and biceps weakness implies a lesion affecting the upper trunk. More distally, the lateral cord gives rise to the lateral pectoral nerve; the posterior cord gives rise to the upper subscapular nerve, the

**Table 5–1.** Nerves and Muscles of the Brachial Plexus 

<b>Upper Limb</b>		<b>Spinal Roots</b>
Spinal Accessory Nerve		
Trapezius		C-3, C-4
Brachial Plexus		
Rhomboids		C-4, C-5
Serratus anterior		C-5, C-6, C-7
Pectoralis major		
Clavicular		C-5, C-6
Sternal		C-6, C-7, C-8
Supraspinatus		C-5, C-6
Infraspinatus		C-5, C-6
Latissimus dorsi		C-6, C-7, C-8
Teres major		C-5, C-6, C-7
Axillary Nerve		
Deltoid		C-5, C-6
Musculocutaneous Nerve		
Biceps		C-5, C-6
Brachialis		C-5, C-6
Radial Nerve		
Long head		
Triceps	Lateral head	C-6, C-7, C-8
Medial head		
Brachioradialis		C-5, C-6
Extensor carpi radialis longus		C-5, C-6
Posterior Interosseous Nerve		
Supinator		C-6, C-7
Extensor carpi ulnaris		C-7, C-8
Extensor digitorum		C-7, C-8
Abductor pollicis longus		C-7, C-8

(continued)

**Table 5–1.** Nerves and Muscles of the Brachial Plexus (*continued*)

Upper Limb	Spinal Roots
Extensor pollicis longus	C-7, C-8
Extensor pollicis brevis	C-7, C-8
Extensor indicis	C-7, C-8
Median Nerve	
Pronator teres	C-6, C-7
Flexor carpi radialis	C-6, C-7
Flexor digitorum superficialis	C-7, C-8, T-1
Abductor pollicis brevis	C-8, T-1
Flexor pollicis brevis	C-8, T-1
Opponens pollicis	C-8, T-1
Lumbricals I & II	C-8, T-1
Anterior Interosseous Nerve	
Pronator quadratus	C-7, C-8
Flexor digitorum profundus I & II	C-7, C-8
Flexor pollicis longus	C-7, C-8
Ulnar Nerve	
Flexor carpi ulnaris	C-7, C-8, T-1
Flexor digitorum profundus III & IV	C-7, C-8
Hypothenar muscles	C-8, T-1
Adductor pollicis brevis	C-8, T-1
Flexor pollicis brevis	C-8, T-1
Palmar interossei	C-8, T-1
Dorsal interossei	C-8, T-1
Lumbricals III & IV	C-8, T-1

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thoracodorsal nerve, and the lower subscapular nerve; and the medial cord gives rise to the medial pectoral nerve, the medial brachial cutaneous nerve, and the medial antebrachial cutaneous nerve. Again, lack of function of the muscles innervated by these nerves can be used to determine the site of injury within the brachial plexus.

### Proximal and Distal Anatomic Relationships

The dorsal rootlet (sensory) and ventral rootlet (motor) converge to form a spinal nerve root. These two structures converge near the neural foramen (Fig. 5-2; see color plate). The cell bodies of the axons of the sensory rootlet reside in the dorsal root ganglion (outside of the spinal cord), whereas cell bodies of the motor rootlet lie within the anterior horn of the spinal cord. The nerve root is enveloped by the epineurium, which is confluent with the dura; however, the epineurium is not attached to the foramen.

As the spinal nerves emerge from the neural foramina, they receive contributions from the sympathetic ganglia (Fig. 5-2; see color plate). Typically, the C-5 and C-6 nerves receive contributions from the middle cervical ganglion, C-7 and C-8 nerves receive contributions from the inferior cervical ganglion, and the T-1 nerve receives a contribution from its associated ganglion. Understanding the relationship of the brachial plexus to the sympathetic ganglia allows the examiner to understand the association of a proximal C-8 and T-1 lesion with the presence of ipsilateral Horner syndrome (loss of sympathetic function of the eye manifested as ptosis, miosis, and anhidrosis).

The nerve roots of C-5 through C-7 emerge from the vertebral foramina and separate into anterior (innervates the upper extremity) and posterior (innervates the paraspinal muscles and posterior vertebral elements) rami. The anterior rami lie in a groove in the transverse process that is posterior to the vertebral artery (Fig. 5-3; see color plate). The anterior rami usually emerge between the anterior and middle scalene muscles to form the upper trunk. The nerve roots of C-8 and T-1 are retroclavicular. The first and second rib lie posterior, and the pleura lies inferior to these roots; C-8 traverses

superior, and T-1 passes inferior to the first rib. Proceeding distally, the nerve roots join to form the lower trunk on the superior surface of the first rib, which then emerges between the anterior and middle scalene muscles.

The upper, middle, and lower trunks divide into their respective divisions posterior to the clavicle. The divisions progress distally to form cords named in relation to the axillary artery.

### Anatomic Variations

Anatomic variation of the plexus can occur among the spinal roots that contribute to the brachial plexus and can affect the subsequent clinical presentation. Typically, the brachial plexus originates from C-5 to T-1; however, the brachial plexus may receive contributions from C-4 or T-2. Some authors have defined a “prefixed” plexus as one that receives a substantive contribution from C-4 and a “postfixed” plexus as one that receives a substantive contribution from T-2.

There is significant variation from the standard schematic anatomy used to describe the origin of terminal branches of the brachial plexus. In approximately 65% of cases, Ballesteros and colleagues found aberrant origins of the long thoracic, upper subscapular, and lower subscapular nerves (3). Dorsal scapular nerves varied in 50% of cases, and the suprascapular and thoracodorsal nerves had variant origin in approximately 20% of cases.

### Anatomy of Nerve Injury

The nerve roots of the brachial plexus exit from their neural foramina and run along the bony groove between the anterior and posterior tubercles of the vertebrae (Fig. 5-4; see color plate). These bony chutes are well-formed for the nerve roots comprising the upper trunk (C-5 and C-6) and more abbreviated for the nerves comprising the lower trunk. In addition, there is less connective tissue binding the lower nerve roots to the bony chutes when compared with the upper nerve roots. Therefore, the lower nerve roots (C-8 and T-1) are prone to preganglionic injury (avulsion) (Fig. 5-4B; see color plate), whereas the nerves

comprising the upper trunk tend to sustain postganglionic injury (rupture) (Fig. 5-4A; see color plate). A preganglionic injury results in permanent paralysis of the muscles innervated by the avulsed roots and complete sensory loss of the corresponding dermatomes. Spontaneous nerve regeneration is unlikely. A postganglionic injury allows potential retention of function of the cell body within the ventral horn of the spinal cord, and these neurons may regenerate axons under appropriate conditions.

At the microscopic level, Seddon proposed a system for classifying nerve injury in 1943 that is still used today (4). This classification system consists of neurapraxia, axonotmesis, and neurotmesis (Fig. 5-5; see color plate). Neurapraxia generally refers to interruption of the myelin sheath, leaving the axons and surrounding connective tissues intact; this type of injury recovers spontaneously within a few weeks. Axonotmesis refers to interruption of the axons and likely the myelin sheath, but with sparing of the surrounding connective tissues (intact Schwann cell basal lamina); this injury may recover spontaneously, within months to years, only if axonal regeneration is able to progress across the injury zone. Neurotmesis refers to interruption of all elements, including the axons, myelin sheath, and surrounding connective tissues; spontaneous recovery does not occur.

## Clinical Presentation of Brachial Plexus Palsy

The first description of NBPP was reported in the 18th century, and the noticeably shortened weak arm of Kaiser Wilhelm II of Germany has been attributed to NBPP (5). Approximately 1,400 reports regarding NBPP exist in MEDLINE from 1948 to the present, with approximately 10% of those reports appearing within the past year. Improvement in NBPP outcomes has paralleled the increased interest in this condition and may be the result of increased recognition and improved management as well as expanding research through interdisciplinary collaborations.

### Timing of Injury

Determining when an injury to the neonatal brachial plexus occurred may have prognostic as well as medicolegal implications; therefore, the timing should be assessed whenever possible. The medical record should include a careful accounting of the events leading up to delivery of the infant whose course was complicated by shoulder dystocia, as exemplified by the American College of Obstetricians and Gynecologists' Patient Safety Checklist on *Documenting Shoulder Dystocia* (see Appendix C). Regardless of the delivery events, if a neonatal brachial plexus injury is suspected, the site of the injury and its relationship to the shoulder at delivery should be documented. In addition, any neonatal bruising, associated injuries (eg, fractured clavicle), unusual positioning, and presence and location of the caput succedaneum may help elucidate factors that contributed to the surrounding events. Low Apgar scores and abnormal cord blood gas results may serve as markers for reductions in neonatal tone. For some injuries that occurred early in utero, there may be specific physical findings, such as muscle atrophy, contracture, or bone demineralization in the affected extremity. However, absence of these findings does not exclude the possibility of an in utero event.

Efforts have been made to use neonatal electromyography (EMG) testing to determine objectively whether a neonate's injury occurred in utero. The basis for this approach is that muscle fibrillations recorded by EMG represent evidence of denervation that occur 10–14 days after the acute injury. These data however, were generated in the adult patient population. In the neonate, in whom axon lengths are considerably shorter, denervation can be demonstrated much closer to the acute event. Gonik and colleagues examined this relationship in a newborn piglet model and showed that after complete transection of the brachial plexus, denervation was detected by EMG beginning at 24 hours after the experimentally induced injury (6). Although current data do not support the use of delayed newborn EMG testing

to determine when injury occurred, it may have a role with other diagnostic modalities in helping clinicians monitor progression of an injury or decide the timing of surgical intervention. Studies more proximate to the suspected time of injury are needed to better define the future role of EMG in establishing etiologic associations.

### Classification

The most useful classification scheme for clinical presentation of NBPP was proposed by Gilbert and Tassin (7), refined by Narakas (Table 5-2) (8, 9), and supported by Birch (10). Group I represents the clinical findings resulting from nerve injury to C-5 and C-6, characterized by paresis of the deltoid and biceps but active function in limb extensors (elbow, wrist, and hand). The clinical findings in Group II are related to injury of C-5, C-6, and C-7. In addition to paresis of the deltoid and biceps, paresis of the triceps is also seen; however, the long flexors and intrinsic muscles of the hand are relatively unaffected. Group III represents paresis of the entire arm (flail arm) consistent with injury of C-5, C-6, C-7, C-8, and T-1. Group IV manifests as a flail arm with the additional presence of Horner syndrome of the ipsilateral eye, which implies injury to all of the nerve roots of the brachial plexus with a proximal injury to the

lower nerve roots. When this classification system is used between 2 weeks and 4 weeks after birth (when neurapraxic lesions would be recovering), it permits determination of the extent of injury and, more importantly, may guide prognosis.

Other classification schemes are based on the anatomy and physiology of nerve injury. Sunderland reported a physiologic scheme comprising five types of pathology in increasing severity: 1) neurapraxia; 2) axonotmesis; 3) lesion of axon and endoneurium; 4) lesion of axon, endoneurium, and perineurium; and 5) complete transection (11). There also is an anatomic scheme comprising four categories based on anatomic location: 1) upper, 2) intermediate, 3) lower, and 4) total plexus palsy (12, 13). The concept of an upper plexus palsy involving C-5, C-6, and sometimes C-7 was initially defined anatomically by Erb in 1874 (14) after Duchenne described four cases of complete paralysis involving loss of shoulder control and elbow flexion in 1872 (15). Upper plexus palsy, also called Erb palsy, is the most common type of NBPP (10, 16). Erb palsy is visually recognized by the stereotypic “waiter’s tip” posture with the arm adducted, shoulder internally rotated, wrist flexed, and fingers extended. Intermediate palsy was described by Jolly in 1896 (17) and Thomas in 1905 (18), and this condition was thought to involve C-7, C-8, and T-1 nerve roots with the arm

**Table 5–2.** Gilbert and Tassin/Narakas Classification Scheme: Severity of Brachial Plexus Palsy and Prognosis  
 ↵56↵66

Group	Affected nerve roots	Rate of full spontaneous recovery
I	C-5, C-6	~ 90%
II	C-5, C-6, C-7	~ 65%
III	C-5, C-6, C-7, C-8, T-1	< 50%
IV	C-5, C-6, C-7, C-8, T-1 with Horner’s syndrome	~ 0%

Narakas AO. Obstetrical brachial plexus injuries. In: Lamb DW, editor. The paralyzed hand. Edinburgh: Churchill Livingstone; 1987. p. 116–35.

abducted, the elbow flexed, and the fingers and hand flaccid. Lower plexus palsy was described by Dejerine-Klumpke (19, 20); this type of NBPP is rare but can be recognized by a flaccid hand in an otherwise active arm (21). Total plexus palsy is essentially the condition as described by Narakas Groups III and IV, with total loss of function of the arm.

### Assessment of the Infant With Brachial Plexus Palsy

Birth attendants may suspect NBPP on the basis of initial observations of the infant. However, physical examination and ultimate diagnosis are best achieved by the combined efforts of neonatologists, neurologists, pediatricians, physiatrists, and occupational and physical therapists.

The basic premise of the brachial plexus examination relies on an understanding of brachial plexus anatomy. Many of the maneuvers in the physical examination are best evaluated with a patient's voluntary cooperation that neonates are unable to provide. Therefore, different strategies must be used to assess NBPP in neonates compared with older individuals, although the basic anatomic principles remain constant. These strategies also will vary substantially based on the normal development of the infant during the first 2 years of life as motor and sensory functions mature.

A thorough family, maternal, and perinatal history provides context for the physical examination. In the early days after birth, the treating physician should assess the infant for skeletal injuries or fractures by clinical and radiographic examination because some musculoskeletal injuries preclude early therapy for NBPP. No substantial evidence exists to support further injury of the nervous elements with gentle handling of the neck and affected limb, and immobilization is not recommended except when associated with skeletal injuries. Surveillance of spontaneous movements and normal reflexes should be performed because global neurologic deficits may indicate other neurologic disorders that can occur concur-

rently with NBPP (22). Similarly, the presence of ptosis and meiosis, consistent with Horner syndrome, indicates lower trunk involvement. An observed asymmetric expansion of the chest cavity and difficulty with oxygenation or feeding can indicate diaphragmatic palsy resulting from phrenic nerve injury, which can be confirmed with plain radiographs, ultrasonography, or fluoroscopy even in the intubated patient. Diaphragmatic palsy can be a dangerous condition resulting in respiratory distress and early failure to thrive and should be addressed promptly (23). Likewise, observation of classic postures (eg, waiter's tip) implies particular NBPP lesions.

With regard to specific motor function and localization of the NBPP lesion, the treating physician should assess the passive and active range of motion of the affected arm. Because contractures and joint subluxations do not develop until several months after birth, early limitations of passive range of motion imply other musculoskeletal disorders or in utero onset of peripheral nerve injury (24, 25). Active range of motion and muscle power can be difficult to assess because infants do not follow commands, but engaging the neonate with stimulation, including with irritating stimuli, can be instructive. Significant motor and sensory function can be gleaned from spontaneous or stimulated responses. Sensory function is similarly difficult to assess in detail, but an overall impression can be inferred by judging the infant's response to particular stimuli (eg, pinprick, pinch, heat, or cold). Indications of chewing or biting of the arm or hand imply sensory alterations in the affected area (26). The presence of trophic skin changes in dermatomal distributions can also indicate sensory alterations. As the infant grows, measurements of the circumference and length of the arm can be tracked as indicators of musculoskeletal dysfunction (27).

Supplementing the physical examination with radiographic and electrodiagnostic findings is helpful to decide whether nerve repair or reconstruction will be beneficial, and detailed discussions regarding these topics are found elsewhere (28, 29). Early referral of those with NBPP and

extensive nerve injury may improve outcomes: for example, the early presence or absence of elbow extension or elbow flexion and of motor unit potentials (electrodiagnostic findings) in the biceps muscle correctly predicted whether lesions were mild or severe with respect to long-term involvement in 85–94% of infants (30).

### Assessment Scales

#### Assessment of Motor Function

Assessment scales in NBPP are used to gauge the extent of injury, prognosticate potential recovery, and determine further treatment. These scales generally focus on joint angles or muscle activation. Muscle power is generally expressed through the U.K. Medical Research Council Scale for muscle movement (MRC scale) (Table 5-3). This scale provides structured grading of individual muscle groups but does not provide any information about overall function of the limb or child. Because the MRC scale requires voluntary cooperation, it is not reliably applied in newborns but can be estimated from observation of the infant's responses to stimulation (31). To overcome these difficulties in assessing the motor function in newborns, Curtis and colleagues proposed the Active Movement Scale (AMS) (Table 5-4) (32). In two complementary studies, the AMS was reported to be a reliable tool for evaluating infants with upper-extremity paresis, and its inter-rater variability demonstrated reliability independent of the rater experience.

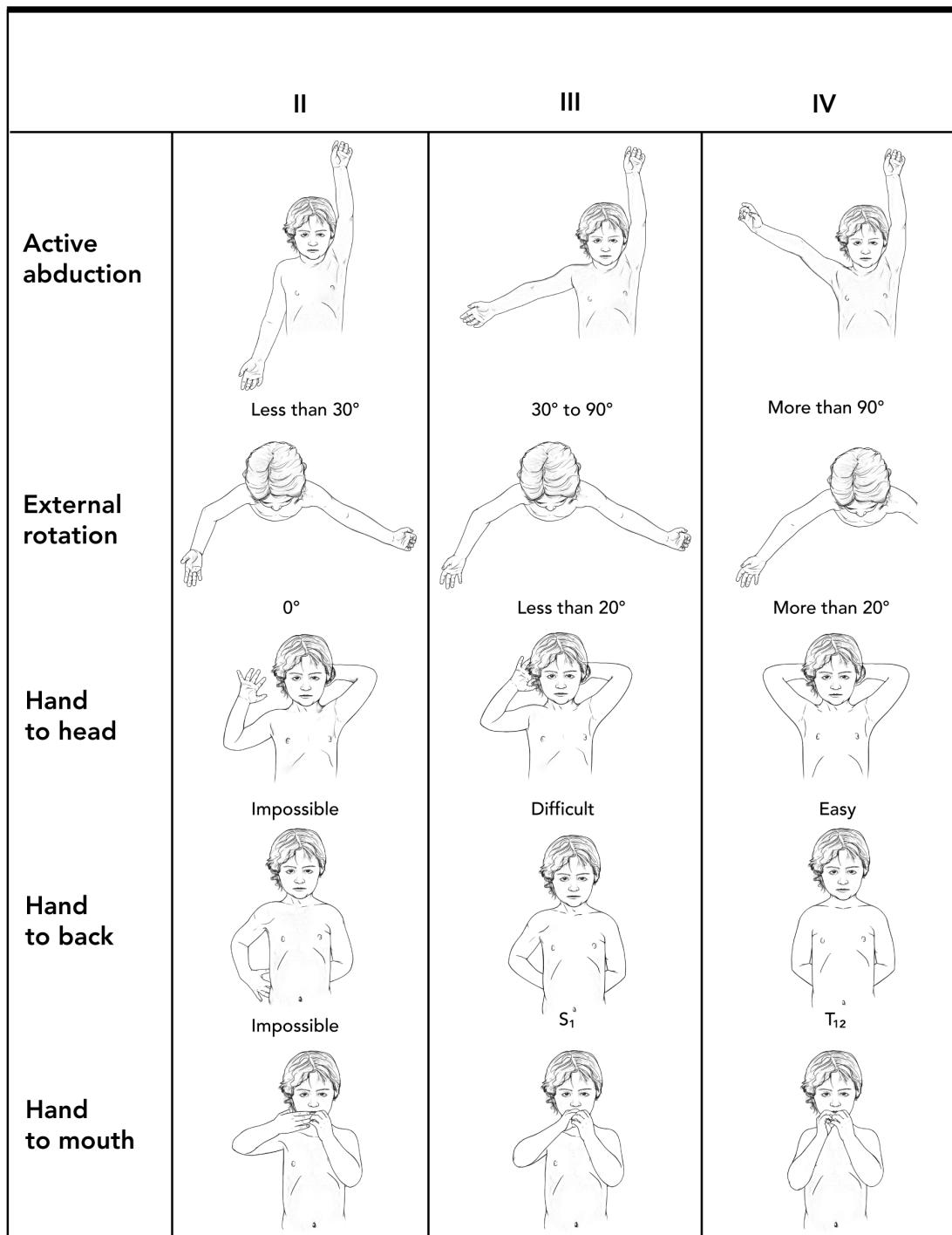
As the infant grows, the function of the whole limb becomes critical. The recovery of shoulder movements may be incomplete after NBPP, and the Mallet scale provides a quantifiable assessment for shoulder function (Fig. 5-6) (33). The Mallet scale can be used in conjunction with Gilbert's classification of shoulder paralysis (Table 5-5), with some consistency reported between the two systems (10). The movements assessed in the Mallet scale focus on the shoulder and elbow, so it primarily addresses upper plexus function in older children because it requires cooperation of the child. Nonetheless, some items (hand-to-mouth, presence of trumpet sign, and lateral reach to assess shoulder abduction) can be used to estimate shoulder function even in infants. For elbow function, an elbow recovery scale has been suggested by Gilbert and Raimondi (Table 5-6) (34). Similarly, Raimondi has proposed a hand evaluation scale (Table 5-7) which has been used to assess hand function after nerve repair or reconstruction and found to correlate with the preoperative Gilbert and Tassin/Narakas group (35, 36).

#### Assessment of Overall Function

Although the previously mentioned assessment scales have been the mainstay of outcomes reporting by surgeons as a measure of technical success or for preoperative evaluation (37–39), they do little to address the overall function of the child (40). A scheme based on five dimensions of disablement

**Table 5-3.** United Kingdom Medical Research Council Scale for Muscle Movement and Power ⇤

Grade	Effort
M0	No detectable muscle contraction
M1	Palpable muscle contraction without movement
M2	Movement in a horizontal plane
M3	Movement overcoming the pull of gravity
M4	Movement overcoming resistance beyond the pull of gravity
M5	Normal strength

**Figure 5-6.** The Mallet classification for assessing shoulder function.

Grade I - No function

Grade V - Normal Function ↵

**Table 5–4.** The Active Movement Scale for Assessing Motor Function in Newborns ↵

Observation	Muscle grade
Gravity eliminated	
No contraction	0
Contraction, no motion	1
Motion less than or equal to $\frac{1}{2}$ range	2
Motion greater than $\frac{1}{2}$ range	3
Full motion	4
Against gravity	
Motion less than or equal to $\frac{1}{2}$ range	5
Motion greater than $\frac{1}{2}$ range	6
Full motion	7

Curtis C, Stephens D, Clarke HM, Andrews D. The active movement scale: an evaluative tool for infants with obstetrical brachial plexus palsy. *J Hand Surg Am* 2002;27:470–8.

**Table 5–5.** Gilbert Scale for Staging Shoulder Function ↵

Shoulder Functions	Stage
Flail shoulder	0
Abduction or flexion to 45 degrees; no active external	I
Abduction less than 90 degrees; external rotation to neutral	II
Abduction=90 degrees; weak external rotation	III
Abduction less than 120 degrees; incomplete external rotation	IV
Abduction greater than 120 degrees; active external rotation	V
Normal	VI

Birch R, Bonney G, Wynn Parry CB. Birth lesions of the brachial plexus. In: Surgical disorders of the peripheral nerves. London: Churchill Livingstone; 1998. p. 209–33.

**Table 5–6.** Gilbert and Raimondi Scale for Evaluating Elbow Recovery ↵

Elbow Function	Score
Flexion:	
Nil or some contraction	1
Incomplete flexion	2
Complete flexion	3
Extension:	
No extension	0
Weak extension	1
Good extension	2
Extension Deficit:	
0–30 degrees	0
30–50 degrees	-1
Greater than 50 degrees	-2

Gilbert A, Raimondi P. Evaluation of results in obstetric brachial plexus palsy. The elbow. International meeting on obstetric brachial plexus palsy. Heerlen, NL; 1996.

**Table 5–7.** Raimondi Scale for Evaluating Hand Function ↵

Description	Grade
Complete paralysis or slight finger flexion of no use; useless thumb—no pinch; some or no sensation	0
Limited active flexion of fingers; no extension of wrist or fingers; possibility of thumb lateral pinch	I
Active flexion of wrist, with passive flexion of fingers (tenodesis); passive lateral pinch of thumb	II
Active complete flexion of wrist and fingers; mobile thumb with partial abduction–opposition. Intrinsic balance; no active supination; good possibilities for palliative surgery	III

(continued)

**Table 5–7.** Raimondi Scale for Evaluating Hand Function (continued)

Description	Grade
Active complete flexion of wrist and fingers; active wrist extension; weak or absent finger extension. Good thumb opposition, with active ulnaris intrinsics; partial pronation/supination	IV
Hand IV, with finger extension and almost complete pronation/supination	V

Raimondi P. Evaluation of results in obstetric brachial plexus palsy. The hand. International meeting on obstetric brachial plexus palsy. Heerlen, NL; 1993.

proposed by the National Center for Medical Rehabilitation Research comprises 1) pathophysiology, 2) impairment, 3) functional limitation (activity), 4) disability (participation), and 5) societal limitation (41). It demonstrates well the shortcomings of the other assessment scales described, especially regarding the functional limitation (activity), disability (participation), and societal limitation in those with NBPP. Likewise, the International Classification of Functioning, Disability, and Health defines function based on body functions, activities, and participation. Also, Boeschoten and colleagues reported on an approach in which a defined set of activities was assessed by videotape and scored; the approach shows potential for assessing the functional activities of children with NBPP, but a number of difficulties remain in observing and scoring using this method (42). Speech dominance (43) and limb preference (44) also have been studied in the context of NBPP. Sundholm and colleagues contend that NBPP should be described in terms of impairment and disability; they reported that many children had difficulty with activities of daily living (45).

Another assessment of global function of the child with NBPP uses the Pediatric Outcomes Data Collection Instrument (PODCI) (46). This assessment obtains semiquantitative, patient-reported,

and parent-reported measures of function and quality of life with respect to several domains: mobility and transfers, upper extremity function, ability to participate in sports, comfort or pain, and happiness. With regard to NBPP, the PODCI was shown to differentiate reliably those children who were potential candidates for reconstructive surgery from children without a musculoskeletal disorder (47). The PODCI also has been used to evaluate the results of tendon transfers for shoulder external rotation in children with NBPP. A 2009 report showed that despite lower PODCI scores, children with NBPP safely participated in a variety of sports at levels similar to their peers (48).

With further regard to self-care and activities of daily living in the child with NBPP, the Pediatric Evaluation of Disability Inventory (PEDI) is a tool used to determine a child's ability to perform self-care activities in relation to developmental age-expected performance (49). The PEDI was unable to discriminate between the self-care ability of children with NBPP versus their peers but was effective in distinguishing between the different levels of NBPP severity (50). Application of PODCI and PEDI are important steps toward functional assessments for determining later childhood treatment and for evaluating treatment efficacy in patients with NBPP.

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# Chapter 6

## Patterns of Neonatal Brachial Plexus Palsy and Outcomes

### Incidence of Brachial Plexus Palsy Patterns

Among cases of neonatal brachial plexus palsy (NBPP), the upper plexus is affected more frequently (Erb palsy) than either the lower plexus alone (classic Klumpke palsy) or the complete nerve complex. In a review of 87 children with residual NBPP at 2 months of age, Wickstrom reported that 54 (62%) displayed an injury to the C-5 and C-6 region of the plexus, 11 (13%) showed evidence of damage to only the C-8 and T-1 region of the plexus, and 22 (25%) had complete residual palsies (1). Wickstrom also was one of the first researchers to note the correlation between the severity of the injury, in terms of long-term improvement, and the level of involvement. Seventy-eight percent of the children in his case review who were classified as having an upper plexus injury had substantial, although not necessarily complete, recovery by 6 months of age. In comparison, only 3 of 22 children (14%) with complete plexus involvement and 1 of 11 (9%) of those with lower plexus injuries were classified as having a minimal injury and had experienced significant recovery without surgical intervention.

### Natural History

The natural history of NBPP, around which the determination of optimal treatment revolves, remains the subject of speculation and debate in many published reports. The complete potential scope of NBPP is difficult to define because of the various combinations of lesions within the elements of the brachial plexus. For example, because the brachial plexus is composed of five roots, three trunks, six divisions, three cords, and five terminal branches (see anatomy description in Chapter 5), thousands of theoretically different brachial plexus lesions are possible for the nerve injuries alone, even without regard to additional musculoskeletal issues. Further difficulties include the definition of recovery and the potential bias introduced by the referral patterns of reporting physicians (1, 2) because many patients with Erb palsy recover spontaneously and are not referred to the specialists who publish most reports. With these caveats in mind, some authors provide an encouraging view of the natural history of NBPP, with a more than 80% occurrence of a favorable outcome or complete recovery (3–8), whereas other authors provide a

contrasting view, with less than 50% of patients demonstrating good recovery or freedom from persistent disabilities (2, 9–13). A 2009 study by Foad and colleagues addressed the discrepancy between the spontaneous recovery rate of NBPP often cited as 75–95% and recent reports finding much lower recovery rates. Across 11 studies with recovery of function described at 3 months and 6 months, 64% of infants classified as C-5–C-6 or C-5–C-6–C-7 had spontaneous recovery of biceps function at age 3 months and complete recovery at 6 months, in comparison to 9% of C-5–T-1 (with or without Horner syndrome) with recovery at 3 months and 14% at 6 months. Regardless of the neurologic recovery, functional recovery also can be compromised by musculoskeletal defects (eg, contractures or joint subluxation), even with appropriate therapy (14).

Most practitioners agree that as the absence of spontaneous clinical improvement persists with increasing time, the potential for recovery diminishes (4, 15, 16). A detailed prospective study by Gilbert and Tassin reported that 32% of a cohort of patients with NBPP made a complete recovery (17). These patients were characterized by early rapid improvements in arm function with recovery of deltoid and biceps function before 2 months of age. Forty-three percent of this cohort made less than full recovery. This group of patients was characterized by slow progress, with no evidence of biceps recovery until after 6 months of age. The study by Gilbert and Tassin led to several prognostic conclusions: 90% of patients with Group I NBPP (C-5–C-6) progressed to full spontaneous recovery if there were clinical signs of recovery before 2 months of age. Approximately 65% of patients with Group II (C-5–C-6–C-7) palsy recovered fully, but the remainder had persistent deficits in shoulder and elbow movement. The timing of recovery of this group of patients was delayed, with clinical signs of recovery not evident until 3–6 months of age. For those in Group III (C-5–C-6–C-7–C-8–T-1, without Horner syndrome), less than 50% recovered fully spontaneously, with the majority having deficits of movement throughout

the arm; in approximately 25% of those studied in this group, even wrist and finger extension remained functionally compromised. Those patients with Group IV NBPP (C-5–C-6–C-7–C-8–T-1, with Horner syndrome) had little chance for a full spontaneous recovery. The classification and prognostication system attributed to Gilbert, Tassin, and Narakas remains a popular classification system not only for its ability to describe the severity of the pathology, but also for its capacity to provide prognostic information (see Chapter 5, Table 5-2) (18).

Most practitioners agree that early recovery is associated with favorable outcomes (19). The Collaborative Perinatal Study reported that 93% of patients who went on to full spontaneous recovery had done so by 4 months of age (4). Metaizeau and colleagues reported that patients who showed no signs of clinical improvement by 3 months did not recover function adequately, and those who did not show improvement by 6 months had essentially no chance of adequate functional recovery (20). Bennet and Harrold reported that infants who recovered fully began to show clinical signs of improvement by 2 weeks of age (3). Yet other authors contend that all infants who recover satisfactorily achieve biceps and deltoid function by 3 months of age (21, 22). Similarly, failure to recover antigravity power in the proximal muscle groups by 6 months of age predicts future moderate-to-severe weakness in the affected extremity (23, 24). Fisher and colleagues, however, noted that early elbow flexion alone is likely not a sufficient criterion to recommend for or against nerve repair or reconstruction (25).

The predictors of recovery described use simple clinical muscle assessments. Some authors have constructed paradigms based on more complicated statistical analyses of multiple independent clinical variables (5, 26). These models, however, have not shown clinically significant improvement in predicting recovery over the simpler indicators. A number of children appear normal and seem to have recovered function, but the affected extremity is not equally functional, as measured by very sensitive tests.

## Operative Outcomes

The indications for surgical nerve reconstruction in NBPP vary widely among different practitioners, with the exception of Narakas Group III and IV lesions, for which nerve reconstruction is generally recommended. For example, the inability to pass the “cookie test” (the ability of babies to bring a cookie to their mouths with the affected limb) at 9 months of age has been reported as a reasonable indication for surgery (27, 28). Bertelli suggests the “towel test”—NBPP patients who fail to remove a towel covering their faces at 6 months are referred for nerve reconstruction (29). Gilbert recommends nerve reconstruction for infants who have not recovered biceps function after 3 months (30). Many practitioners use a combination of these assessments and ancillary studies to guide their practice, but standard guidelines have yet to be developed.

Similarly, many difficulties (including the variability of anatomic lesions in the complex brachial plexus structure and adjacent musculoskeletal elements, differing surgical strategies, and varied assessment techniques) preclude direct comparison of surgical results among studies. Therefore, the following describes the results from representative studies. As discussed by Lin and colleagues, early improvements in neurologic function produced by neurolysis in NBPP were unsustained over time, whereas nerve grafting after resection of the neuroma-in-continuity produced significant improvements in function with a mean follow-up of 4 years (31). Shenaq and colleagues reported that in 282 infants with NBPP with a mean follow-up of 5 years, 75% had good to excellent results after primary and secondary reconstructive surgery (32). Gilbert and colleagues reported the results for 436 patients who underwent nerve reconstruction and secondary reconstructive procedures for NBPP. Eighty percent with Group I palsy and 61% with Group II palsy had “good” or “excellent” shoulder function after 4 years (30, 33). For elbow function, the study reported “good” results in all of the Group I and Group II patients and “good” results in 81% of Group III and Group IV patients.

Birch and colleagues reported the outcome of surgical treatment of the NBPP by nerve root lesion repairs (34). For C-5 repairs, 33% had a “good” result and 48% had “fair” results for recovering shoulder function; 55% of repaired C-6 lesions resulted in a “good” outcome, and 26% had a “fair” outcome. For C-7 root repairs, mostly “poor” results were obtained, with only 24% of patients achieving full wrist extension. In patients with C-8–T-1 lesions, 57% of the patients achieved “good” results and 36% achieved “fair” results after nerve reconstruction. In contrast, Haerle and colleagues published the results of 98 patients with total plexus palsy (Group III and Group IV) who underwent nerve reconstruction and secondary reconstructive surgery (35). Two years following primary nerve surgery alone, the results at the shoulder were “poor” for 75%, which may have been the result of the surgical strategy and priority of function in these patients with total lack of arm function. However, after the secondary shoulder reconstructive procedures, the results improved, with 44% having “good” or “excellent” function at the shoulder. For elbow function, 68% had a “good” or “excellent” elbow flexion after nerve reconstruction alone, but 81% had “good” elbow flexion after additional secondary reconstruction. For hand function, 35% of children had useful hand function after nerve reconstruction alone, but 76% had useful hand function after the additional secondary procedures. These results reflect the complex nature of the surgical strategy and the need to evaluate the treatment course longitudinally, even after a primary surgery is completed.

Additionally, nerve transfers are often undertaken as part of the nerve reconstruction or alone as an isolated procedure in adults, but the use of nerve transfers may be increasing in infants. The spinal accessory nerve has been used widely to neurotize the suprascapular nerve to improve shoulder function by reinnervating the supraspinatus and infraspinatus muscles (part of the rotator cuff). The results of this transfer are difficult to assess because it is often done in conjunction with other nerve transfers to restore deltoid muscle func-

tion. Terzis and colleagues reported significantly better functional outcomes for supraspinatus and infraspinatus muscles in infants who had reconstruction within the first 6 months of life (36). In contrast, Pondaag and colleagues reported less satisfying results for infraspinatus function after a combined nerve repair from C-5 to the suprascapular nerve and nerve transfer from the spinal accessory to the suprascapular nerve, with only 20% of patients demonstrating more than 20 degrees of external rotation (37). Note that only a small degree of external rotation is required for adequate function at the shoulder; 75% of these patients were able to reach the back of the head.

Nerve transfers to restore elbow flexion generally yield good to excellent results. For example, intercostal nerves as donor nerves in nerve transfer strategies to the musculocutaneous nerve provide functional elbow flexion (37–40). Likewise, Al-Qattan (41) and Noaman and colleagues (42) reported on patients who recovered essentially normal biceps function several months after nerve transfer from ulnar motor fascicles to the musculocutaneous nerve. Wellons and colleagues reported that 80% gained functional elbow flexion after medial pectoral to musculocutaneous nerve transfer (43).

## Nonoperative Outcomes

The majority of infants with NBPP with injuries that do not resolve spontaneously are managed nonoperatively (44). Physical therapy or occupational therapy to maintain range of motion and to strengthen and facilitate function and awareness of the limb while waiting for reinnervation, are indicated from the first weeks of life when there is persisting weakness (45–47). Use of neuromuscular electrical stimulation (NMES) is used in the NBPP population as a method of strengthening (46) in young children who may otherwise be unable to fully cooperate. Using kinesio tape also is common to facilitate active movement. Aquatic therapy offers an antigravity environment and may facilitate emerging recovery of movement. Splints may be indicated to maintain joint flexibility while waiting

for reinnervation. Programs of activities and exercises provided for infants and children with NBPP should be age-appropriate and inclusive of a home program. Therapy is indicated with or without surgical interventions for those children with functional impairment (46). Postoperatively, NMES is used for strengthening emerging movement after nerve grafting or transfers and for neuromuscular reeducation and strengthening of transferred muscles to perform new movements, sometimes antagonistic to those performed before the insertion. Participation in preferred age-appropriate typical activities that incorporate movement of the affected limb also should be encouraged.

Botulinum toxin type A injections have been shown in several small studies to be effective for muscle imbalance (48–51), cocontractions (52–54), and contractures (50, 55) at affected joints in the upper extremity in children with NBPP. Botulinum toxin type A temporarily blocks neuromuscular transmission by inhibiting the release of acetylcholine and can be used to temporarily weaken the stronger unaffected antagonistic muscles, and to potentially allow the strengthening and emergence of function of the weaker affected agonists (56). Although use of botulinum toxin type A in NBPP is off-label, its safety profile and efficacy have been well established (56).

Prospective randomized controlled trials evaluating the effectiveness of either surgical or rehabilitative interventions after NBPP have not been conducted; the literature does nonetheless support these interventions. In the event that there is no spontaneous resolution, children with NBPP will benefit from timely referrals for operative or nonoperative intervention to minimize the consequences of the injury.

## Natural Course of Neonatal Brachial Plexus Palsy With and Without Shoulder Dystocia

In 1998, Gherman and colleagues examined the difference in persistence of NBPP that occurred in the presence of shoulder dystocia with those that showed no evidence of shoulder dystocia

(57). Of the 17 injuries documented without shoulder dystocia, the average time for the injury to resolve was significantly longer than those in whom shoulder dystocia occurred (6 months versus 2 months). In addition, a higher number of NBPP cases without shoulder dystocia were persistent at 1 year of life (41% versus 8%).

## Summary

Overall, infants who sustain NBPP have a good prognosis, with the majority recovering adequate functional use of the affected arm without surgical intervention. Early occupational therapy and physical therapy can support spontaneous recovery of function and minimize musculoskeletal comorbidities. Given recent and continuing improvements in surgical and conservative treatments, early referral to a specialty care center can improve overall outcomes after NBPP.

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# Glossary

**Axial traction:** A pulling force (traction) applied in alignment with the fetal cervicothoracic spine, sometimes referred to as “downward axial traction.”

**Axonotmesis:** A more severe grade of nerve injury, characterized by interruption of the axons (and likely adjacent myelin) with preservation of the surrounding connective tissues that theoretically can support axonal regeneration. Note that the nerve distal to the injured segment must undergo Wallerian degeneration prior to nerve regeneration.

**Biofidelity:** The quality of being lifelike in appearance or response, as in test dummies for safety procedures.

**Brachial plexus:** A network of spinal nerves that originates in the spinal cord, extends through the axilla, and gives rise to nerves to the upper limb, carrying motor, sensory, and autonomic nervous system elements.

**Clinical shoulder dystocia:** Failure of delivery of the fetal shoulders diagnosed when attempts made with gentle downward traction have been unsuccessful in delivering the shoulders.

**Cocontraction:** The simultaneous contraction of agonist and antagonist muscles around a joint to hold a position.

**Compression:** A pushing force that acts to shorten or compact (squash) an object.

**Diagnostic traction:** A pulling force (traction) required to make the initial diagnosis of shoulder dystocia or used to determine whether an ancillary obstetric maneuver has successfully alleviated the shoulder dystocia.

**Endogenous force:** Expulsion force generated by uterine contractions and maternal pushing efforts.

**Erb palsy:** A form of brachial plexus palsy that manifests as paralysis of the muscles of the upper arm and shoulder girdle due to an injury to the fifth and sixth (and/or seventh) cervical nerve roots or the upper and middle part of the brachial plexus.

**Excessive traction:** A pulling force (traction) that is greater than the inherent strength of the fetal brachial plexus.

**Exogenous force:** Extraction force applied to the fetal presenting part by the delivering practitioner during vaginal (or cesarean) delivery.

**Failure force (or failure load):** The amount of applied force that causes a structure to fracture or rupture, measured in units of Newtons (N) or pounds of force (lbf).

**Fundal pressure:** External pressure applied through the anterior abdominal wall to the top of the uterus.

**Gaskin maneuver:** A technique in which the patient assumes a position on her hands and knees (“all fours”) in order to facilitate a delivery.

**High-fidelity simulation:** A simulation technique in which realistic materials or models are used and trainees are provided with data, instrument readings, or other observable phenomena to evaluate complex physical responses.

**Klumpke palsy:** A form of brachial plexus palsy that manifests as paralysis of the muscles of the medial forearm and hand due to an injury to the eighth cervical and first thoracic nerve roots or the lower part of the brachial plexus.

**Lateral traction:** A pulling force (traction) that also bends a fetus’ neck by moving the ear toward the posterior shoulder, thereby opening the contralateral angle of the neck and increasing strain on the brachial plexus, sometimes referred to as “lateral bending,” “downward lateral bending,” or “downward lateral traction.”

**Low-fidelity simulation:** A simulation technique involving simple signal modeling that attempts to replicate reality and is usually used for training in specific tasks.

**Macrosomia:** Growth beyond a specific birth weight, usually 4,000 g, regardless of the gestational age.

**Maternal explosive force:** A force generated by the gravid patient through uterine contractions and/or maternal pushing.

**McRoberts maneuver:** A technique in which, from the lithotomy position, the maternal thighs are placed in a position of exaggerated hyperflexion and abduction.

**Nerve avulsion:** Tear of a spinal nerve root away from the spinal cord, within or proximal to the vertebral foramen.

**Nerve rupture:** Tear of a spinal peripheral nerve root at a point distal to the vertebral foramen.

**Neurapraxia:** The mildest grade of nerve injury resulting in reduced or absence of conduction across the injured segment of nerve that may be due to interruption of the myelin sheath or metabolic abnormalities. Note that axonal continuity is maintained.

**Neuroma:** Disorganized mass of nerve fibers and connective tissue that can be the normal response of a nerve to injury.

**Neurotmesis:** The most severe grade of peripheral nerve injury with complete interruption of the axon, myelin, and connective tissues. Note that recovery and regeneration cannot occur spontaneously.

**Newton:** Metric unit of force, approximately equal to one fourth of a pound of force (lbf).

**Postfixed brachial plexus:** Anatomic variant of the brachial plexus in which the second thoracic nerve root contributes to the brachial plexus.

**Postganglionic nerve injury (rupture):** Injury to the nerve root and rootlets distal to the dorsal root ganglion.

**Prague maneuver:** A method for delivering a fetus in breech position in which the infant's shoulders are grasped from below by one hand while the other hand supports the legs.

**Prefixed brachial plexus:** Anatomic variant of the brachial plexus in which the fourth cervical nerve root contributes to the brachial plexus.

**Preganglionic nerve injury (avulsion):** Injury to the nerve root and rootlets proximal to the dorsal root ganglion.

**Rubin maneuver:** A technique in which the practitioner applies pressure to the posterior surface of either the posterior or anterior fetal shoulder, toward the fetal chest.

**Strain:** Engineering term describing the amount of deformation in an object compared with its original length (change in length). Strain can be positive (resulting from traction) or negative (resulting from compression).

**Suprapubic pressure:** Pressure that is applied to the lower abdomen above the symphysis pubis. It can be either directed posteriorly (downward), with lateral application from either side of the maternal abdomen (laterally or obliquely), or alternating between sides using a rocking movement. Pressure is ideally applied from the side of the fetal back.

**Symphiotomy:** A surgical technique involving incision of the connective tissue joining the anterior rami of the symphysis pubis.

**Tensile load:** The amount of traction force (tension) that is applied to a structure, measured in units of Newtons (N) or pounds of force (lbf).

**Traction:** A pulling force that acts to elongate or stretch an object.

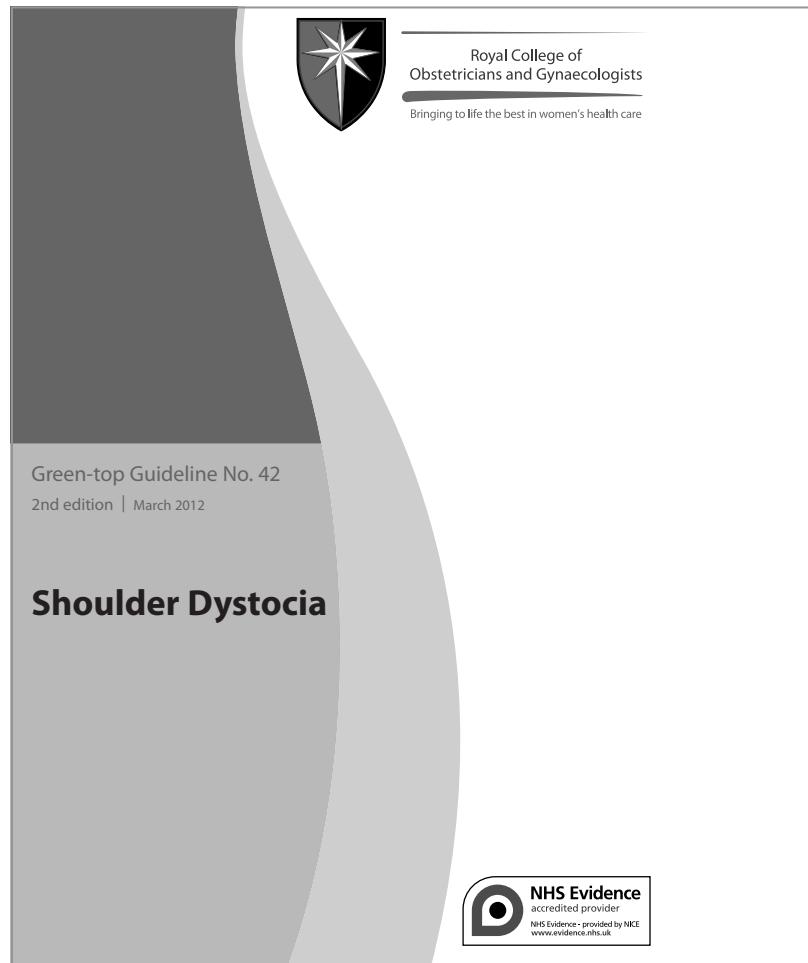
**Turtle sign:** The retraction of the fetal head against the maternal perineum, accompanied by difficulty in accomplishing external rotation.

**Wood maneuver:** A technique in which the birthing attendant attempts to rotate the fetus by exerting pressure on the anterior surface of the posterior shoulder, toward the fetal back.

**Zavanelli maneuver:** A technique that requires manual replacement of the fetal vertex into the vagina, followed by cesarean delivery.

# Appendix A

Royal College of Obstetricians and Gynaecologists  
Green-top Guideline No. 42, *Shoulder Dystocia* ↵



## Shoulder Dystocia

This is the second edition of this guideline. The first edition was published in 2005 under the same title.

### 1. Background

Shoulder dystocia is defined as a vaginal cephalic delivery that requires additional obstetric manoeuvres to deliver the fetus after the head has delivered and gentle traction has failed.<sup>1</sup> An objective diagnosis of a prolongation of head-to-body delivery time of more than 60 seconds has also been proposed<sup>2,3</sup> but these data are not routinely collected. Shoulder dystocia occurs when either the anterior, or less commonly the posterior, fetal shoulder impacts on the maternal symphysis, or sacral promontory, respectively.

There is a wide variation in the reported incidence of shoulder dystocia.<sup>4</sup> Studies involving the largest number of vaginal deliveries (34 800 to 267 228) report incidences between 0.58% and 0.70%.<sup>5-10</sup>

Evidence level  
2+ and Evidence  
level 3

There can be significant perinatal morbidity and mortality associated with the condition, even when it is managed appropriately.<sup>7</sup> Maternal morbidity is increased, particularly the incidence of postpartum haemorrhage (11%) as well as third and fourth-degree perineal tears (3.8%). Their incidences remain unchanged by the number or type of manoeuvres required to effect delivery.<sup>11,12</sup>

Evidence  
level 2+

Brachial plexus injury (BPI) is one of the most important fetal complications of shoulder dystocia, complicating 2.3% to 16% of such deliveries.<sup>7,11,13,14</sup>

Evidence level  
2+ and Evidence  
level 3

Most cases of BPI resolve without permanent disability, with fewer than 10% resulting in permanent neurological dysfunction.<sup>15</sup> In the UK and Ireland, the incidence of BPI was 0.43 per 1000 live births.<sup>16</sup> However, this may be an underestimate as the data were collected by paediatricians, and some babies with early resolution of their BPI might have been missed.

Evidence  
level 2+

There is evidence to suggest that where shoulder dystocia occurs, larger infants are more likely to suffer a permanent BPI after shoulder dystocia.<sup>17,18</sup>

Evidence  
level 4

A retrospective review of all BPIs in one American hospital reported an incidence of 1 in 1000 births, with a permanent injury rate of 0.1 per 1000.<sup>19</sup> Another review of 33 international studies reported an incidence of BPI of 1.4 in 1000 births, with a permanent injury rate of 0.2 per 1000 births.<sup>20</sup>

Neonatal BPI is the most common cause for litigation related to shoulder dystocia and the third most litigated obstetric-related complication in the UK.<sup>21</sup>

Evidence  
level 3

The NHSLA (NHS Litigation Authority) has reported that 46% of the injuries were associated with substandard care.<sup>21</sup> However, they also emphasised that not all injuries are due to excess traction by healthcare professionals, and there is a significant body of evidence suggesting that maternal propulsive force may contribute to some of these injuries.<sup>22,23</sup>

Evidence  
level 3  
and 4

Moreover, a substantial minority of BPIs are not associated with clinically evident shoulder dystocia.<sup>24,25</sup> In one series, 4% of injuries occurred after a caesarean section,<sup>26</sup> and in another series 12% of babies with a BPI were born after an uncomplicated caesarean section.<sup>27</sup> When BPI is discussed legally, it is important to determine whether the affected shoulder was anterior or posterior at the time of delivery, because damage to the plexus of the posterior shoulder is considered unlikely to be due to action by the healthcare professional.<sup>22</sup>

Evidence  
level 4

## 2. Purpose and scope

The purpose of this guideline is to review the current evidence regarding the possible prediction, prevention and management of shoulder dystocia; it does not cover primary prevention of fetal macrosomia associated with gestational diabetes mellitus. The guideline provides guidance for skills training for the management of shoulder dystocia, but the practical manoeuvres are not described in detail. These can be found in the PROMPT (PRactical Obstetric Multi-Professional Training) course manual.<sup>28</sup>

## 3. Identification and assessment of evidence

This RCOG guideline was revised in accordance with standard methodology for producing RCOG Green-top Guidelines. A search was performed in the OVID database, which included Medline, Embase, the Cochrane Database of Systematic Reviews, the Cochrane Control Register of Controlled Trials (CENTRAL), the Database of Abstracts of Reviews and Effects (DARE), the ACP Journal Club, the National Guidelines Clearing House and the Confidential Enquiry into Maternal and Child Health (CEMACH) reports. The search was restricted to articles published between January 1980 and May 2011 and limited to humans and the English language. Search terms included: 'shoulder dystocia', 'macrosomia', 'McRoberts' manoeuvre', 'obstetric manoeuvres', 'complications, labour/delivery', 'brachial plexus injury', 'Erb's palsy', 'Klumpke's palsy', 'symphysiotomy', 'Zavanelli manoeuvre', 'skill drills', 'rehearsal of obstetric emergencies' and 'medical simulation'. Reference lists of the articles identified were hand-searched for additional articles and some experts within the field were contacted. Relevant key original papers published prior to 1980 were also obtained and are referenced within this guideline.

Owing to the emergency nature of the condition, most published series examining procedures for the management of shoulder dystocia are retrospective case series or case reports. Areas lacking evidence are annotated as good practice points.

## 4. Prediction

### 4.1 Can shoulder dystocia be predicted?

**Clinicians should be aware of existing risk factors in labouring women and must always be alert to the possibility of shoulder dystocia.**

D

**Risk assessments for the prediction of shoulder dystocia are insufficiently predictive to allow prevention of the large majority of cases.**

C

A number of antenatal and intrapartum characteristics have been reported to be associated with shoulder dystocia (table 1), but statistical modelling has shown that these risk factors have a low positive predictive value, both singly and in combination.<sup>29,30</sup> Conventional risk factors predicted only 16% of shoulder dystocia that resulted in infant morbidity.<sup>29</sup> There is a relationship between fetal size and shoulder dystocia,<sup>13</sup> but it is not a good predictor: partly because fetal size is difficult to determine accurately, but also because the large majority of infants with a birth weight of  $\geq 4500\text{g}$  do not develop shoulder dystocia.<sup>31</sup> Equally important, 48% of births complicated by shoulder dystocia occur with infants who weigh less than 4000g.<sup>6</sup>

Infants of diabetic mothers have a two- to four-fold increased risk of shoulder dystocia compared with infants of the same birth weight born to non-diabetic mothers.<sup>13,29</sup>

Evidence level  
2+ and Evidence  
level 3

A retrospective case-control study to develop a predictive model of risk for shoulder dystocia with injury was published in 2006.<sup>33</sup> The authors reported that the best combination of variables to identify neonatal injury associated with shoulder dystocia were maternal height and weight, gestational age and parity and birthweight. A score over 0.5 detected 50.7% of the shoulder dystocia cases with BPI, with a false positive

rate of 2.7%.<sup>33</sup> However, the statistical modelling for this prediction tool was based on actual birth weight and not estimated fetal weight. Clinical fetal weight estimation is unreliable and third-trimester ultrasound scans have at least a 10% margin for error for actual birth weight and a sensitivity of just 60% for macrosomia (over 4.5 kg).<sup>34,35</sup> The use of shoulder dystocia prediction models cannot therefore be recommended.<sup>9,35</sup>

**Table 1.** Factors associated with shoulder dystocia

Pre-labour	Intrapartum
Previous shoulder dystocia	Prolonged first stage of labour
Macrosomia >4.5kg	Secondary arrest
Diabetes mellitus	Prolonged second stage of labour
Maternal body mass index >30kg/m <sup>2</sup>	Oxytocin augmentation
Induction of labour	Assisted vaginal delivery

## 5. Prevention of shoulder dystocia

### 5.1 Management of suspected fetal macrosomia

#### 5.1.1 Does induction of labour prevent shoulder dystocia?

**Induction of labour does not prevent shoulder dystocia in non-diabetic women with a suspected macrosomic fetus. Grade D**

D

**Induction of labour at term can reduce the incidence of shoulder dystocia in women with gestational diabetes. Grade B**

B

There are a number of evidence-based reviews that have demonstrated that early induction of labour for women with suspected fetal macrosomia, who do not have gestational diabetes, does not improve either maternal or fetal outcome.<sup>36,37</sup>

Evidence level 4

A systematic review and meta-analysis of randomised controlled trials of the effect of treatment in women with gestational diabetes<sup>38</sup> concluded that the incidence of shoulder dystocia is reduced with early induction of labour.

Evidence level 2+

The NICE diabetes guideline recommends that pregnant women with diabetes who have a normally grown fetus should be offered elective birth through induction of labour, or by elective caesarean section if indicated, after 38 completed weeks.<sup>39</sup>

#### 5.1.2 Should elective caesarean section be recommended for suspected fetal macrosomia to prevent brachial plexus injury (BPI)?

**Elective caesarean section should be considered to reduce the potential morbidity for pregnancies complicated by pre-existing or gestational diabetes, regardless of treatment, with an estimated fetal weight of greater than 4.5 kg.**

D

Infants of diabetic mothers have a two- to four-fold increased risk of shoulder dystocia compared with infants of the same birth weight born to non-diabetic mothers.<sup>15,29</sup> A decision-analysis model estimated that in diabetic women with an EFW > 4.5kg, 443 caesarean sections would need to be performed to prevent one permanent BPI. In comparison, 3695 caesarean sections would be required to prevent one permanent BPI in the non-diabetic population.<sup>34</sup>

Evidence level 3

Estimation of fetal weight is unreliable and the large majority of infants over 4.5kg do not experience shoulder dystocia.<sup>32</sup> In the USA, a decision-analysis model estimated that in non-diabetic women with an EFW of >4kg, an additional 2345 caesarean deliveries would be required, at a cost of US\$4.9 million, to prevent one permanent injury from shoulder dystocia.<sup>34</sup> However, there is some difficulty in grouping all fetuses with an expected weight of >4.5 kg together: some fetuses will be much larger than this. The American College of Obstetricians and Gynecologists (ACOG) has recommended that an estimated fetal weight of over 5 kg should prompt consideration of delivery by caesarean section,<sup>40</sup> inaccuracy of methods of fetal size estimation notwithstanding.

Evidence level 4

The National Institute for Health and Clinical Excellence states that 'ultrasound estimation of fetal size for suspected large-for-gestational-age unborn babies should not be undertaken in a low-risk population'.<sup>41</sup>

### *5.2 What are the recommendations for future pregnancy?*

*What is the appropriate mode of delivery for the woman with a previous episode of shoulder dystocia?*

**Either caesarean section or vaginal delivery can be appropriate after a previous shoulder dystocia. The decision should be made jointly by the woman and her carers.**

**D**

The rate of shoulder dystocia in women who have had a previous shoulder dystocia has been reported to be 10 times higher than the rate in the general population.<sup>42</sup> There is a reported recurrence rate of shoulder dystocia of between 1% and 25%.<sup>5,10,30,42-46</sup> However, this may be an underestimate owing to selection bias, as caesarean section might have been advocated for pregnancies after severe shoulder dystocia, particularly with a neonatal poor outcome.

Evidence level 3

There is no requirement to recommend elective caesarean birth routinely but factors such as the severity of any previous neonatal or maternal injury, predicted fetal size and maternal choice should all be considered and discussed with the woman and her family when making plans for the next delivery.

Evidence level 4

## 6. Management of shoulder dystocia

### *6.1 Preparation in labour: what measures should be taken when shoulder dystocia is anticipated?*

**All birth attendants should be aware of the methods for diagnosing shoulder dystocia and the techniques required to facilitate delivery.**

### *6.2 How is shoulder dystocia diagnosed?*

**Birth attendants should routinely look for the signs of shoulder dystocia.**

Timely management of shoulder dystocia requires prompt recognition. The attendant health carer should routinely observe for:

- difficulty with delivery of the face and chin
- the head remaining tightly applied to the vulva or even retracting (turtle-neck sign)
- failure of restitution of the fetal head
- failure of the shoulders to descend.

**Routine traction in an axial direction can be used to diagnose shoulder dystocia but any other traction should be avoided.**

**D**

Routine traction is defined as 'that traction required for delivery of the shoulders in a normal vaginal delivery where there is no difficulty with the shoulders'.<sup>47</sup> Axial traction is traction in line with the fetal spine i.e. without lateral deviation.

Evidence from cadaver studies suggests that lateral and downward traction, and rapidly applied traction,<sup>48</sup> are more likely to cause nerve avulsion. In a Swedish series, downward traction on the fetal head was strongly associated with obstetric BPI, and had been employed in all cases of residual BPI at 18 months old.<sup>48</sup> Therefore, downward traction on the fetal head should be avoided in the management of all births.

Evidence level 3

There is no evidence that the use of the McRoberts' manoeuvre before delivery of the fetal head prevents shoulder dystocia.<sup>49</sup> Therefore, prophylactic McRoberts' positioning before delivery of the fetal head is not recommended to prevent shoulder dystocia.

### 6.3.1 How should shoulder dystocia be managed?

**Shoulder dystocia should be managed systematically (see appendix 1).**



**Immediately after recognition of shoulder dystocia, additional help should be called.**



**The problem should be stated clearly as 'this is shoulder dystocia' to the arriving team.**



**Fundal pressure should not be used.**



**McRoberts' manoeuvre is a simple, rapid and effective intervention and should be performed first.**



**Suprapubic pressure should be used to improve the effectiveness of the McRoberts' manoeuvre.**



**An episiotomy is not always necessary.**



The Confidential Enquiry into Stillbirths and Deaths in Infancy (CESDI) report on shoulder dystocia identified that 47% of the babies that died did so within five minutes of the head being delivered; however, in a very high proportion of cases, the fetus had a pathological cardiotocograph (CTG) prior to the shoulder dystocia.<sup>50</sup> A group from Hong Kong have recently reported that in their series there was a very low rate of hypoxic ischaemic injury if the head-to-body delivery time was less than five minutes.<sup>51</sup> It is important, therefore, to manage the problem as efficiently as possible to avoid hypoxic acidosis, and as carefully as possible to avoid unnecessary trauma.

Managing shoulder dystocia according to the RCOG algorithm (see appendix 2) has been associated with improved perinatal outcomes.<sup>14</sup>

Evidence level 3

Help should be summoned immediately. In a hospital setting, this should include further midwifery assistance, including the labour ward coordinator or an equivalent experienced midwife, an experienced obstetrician, a neonatal resuscitation team and an anaesthetist.<sup>52</sup>

Evidence level 4

Stating the problem early has been associated with improvements in outcomes in shoulder dystocia<sup>53</sup> and improved performance in other obstetric emergencies.<sup>54</sup>

Evidence level 3

Maternal pushing should be discouraged, as this may exacerbate impaction of the shoulders.<sup>55</sup>

Fundal pressure should not be used during the management of shoulder dystocia.<sup>50</sup> It is associated with a high neonatal complication rate<sup>57</sup> and may result in uterine rupture.<sup>51</sup>

Evidence level 3

The McRoberts' manoeuvre is flexion and abduction of the maternal hips, positioning the maternal thighs on her abdomen.<sup>56</sup> It straightens the lumbosacral angle, rotates the maternal pelvis towards the mother's head and increases the relative anterior-posterior diameter of the pelvis.<sup>57</sup> The McRoberts' manoeuvre is an effective intervention, with reported success rates as high as 90%.<sup>8,11,58,59</sup> It has a low rate of complication and is one of the least invasive manoeuvres, and therefore, if possible, should be employed first.

Evidence level 2+ and Evidence level 3

The woman should be laid flat and any pillows should be removed from under her back. With one assistant on either side, the woman's legs should be hyperflexed. If the woman is in the lithotomy position, her legs will need to be removed from the supports. Routine traction (the same degree of traction applied during a normal delivery) in an axial direction should then be applied to the fetal head to assess whether the shoulders have been released.

If the anterior shoulder is not released with the McRoberts' position and routine axial traction, another manoeuvre should be attempted.

Evidence level 4

Suprapubic pressure can be employed together with the McRoberts' manoeuvre to improve success rates.<sup>11</sup> Suprapubic pressure reduces the fetal bisacromial diameter and rotates the anterior fetal shoulder into the wider oblique pelvic diameter. The shoulder is then freed to slip underneath the symphysis pubis with the aid of routine axial traction.<sup>58</sup>

Suprapubic pressure should ideally be applied by an assistant from the side of the fetal back in a downward and lateral direction just above the maternal symphysis pubis. This reduces the fetal bisacromial diameter by pushing the posterior aspect of the anterior shoulder towards the fetal chest. There is no clear difference in efficacy between continuous pressure and 'rocking' movement. Only routine traction should be applied to the fetal head when assessing whether the manoeuvre has been successful. Again, if the anterior shoulder is not released with suprapubic pressure and routine traction, then another manoeuvre should be attempted.

Evidence level 3

An episiotomy will not relieve the bony obstruction of shoulder dystocia but may be required to allow the healthcare professional more space to facilitate internal vaginal manoeuvres. The use of an episiotomy does not decrease the risk of BPI with shoulder dystocia.<sup>60</sup>

Evidence level 4

An episiotomy should therefore only be considered if internal vaginal access of the healthcare professional's whole hand cannot easily be achieved to facilitate manoeuvres such as delivery of the posterior arm or internal rotation of the shoulders.<sup>61</sup>

### 6.3.2 What measures should be undertaken if simple techniques fail?

**Internal manoeuvres or 'all-fours' position should be used if the McRoberts' manoeuvre and suprapubic pressure fail.**



If simple measures (the McRoberts' manoeuvre and suprapubic pressure) fail, then there is a choice to be made between the all-fours position and internal manipulation.

**Gaining access to the vagina for internal manoeuvres:** the most spacious part of the pelvis is in the sacral hollow; therefore vaginal access should be gained posteriorly, into the sacral hollow. The whole hand should be entered posteriorly to perform internal rotation or delivery of the posterior arm.<sup>62</sup> The woman should be brought to the end of the bed, or the end of the bed should be removed, to make vaginal access easier. Delivery can then be facilitated by rotation into an oblique diameter or when possible by a full 180 degree rotation of the fetal trunk,<sup>63,64</sup> or by delivery of the posterior arm.<sup>65</sup>

Evidence level 4

**Internal rotational manoeuvres** were originally described by Woods<sup>61</sup> and Rubin.<sup>60</sup> Rotation can be most easily achieved by pressing on the anterior or posterior aspect of the posterior shoulder. Pressure on the posterior aspect of the posterior shoulder has the additional benefit of reducing the shoulder diameter by adducting the shoulders.<sup>63</sup> The shoulders should be rotated into the wider oblique diameter, resolving the shoulder dystocia. If pressure on the posterior shoulder is unsuccessful, an attempt should be made to apply pressure on the posterior aspect of the anterior shoulder to adduct and rotate the shoulders into the oblique diameter.

Evidence level 4

**Delivering the posterior arm** reduces the diameter of the fetal shoulders by the width of the arm. The fetal wrist should be grasped and the posterior arm should be gently withdrawn from the vagina in a straight line.<sup>61</sup> Delivery of the posterior arm is associated with humeral fractures with a reported incidence between 2% and 12%,<sup>7,14</sup> but the neonatal trauma may be a reflection of the refractory nature of the case, rather than the procedure itself.<sup>8</sup>

Evidence level 3

There are no randomised comparative studies available comparing delivery of the posterior arm and internal rotation. Some authors favour delivery of the posterior arm over other manoeuvres,<sup>59,66</sup> particularly where the mother is large.<sup>60</sup> Others have reported that rotational methods and posterior arm delivery were similarly successful, but rotational manoeuvres were associated with reductions in both BPI and humeral fractures<sup>68</sup> compared to delivery of the posterior arm. Therefore, healthcare professionals should base their decision on their training, clinical experience and the prevailing circumstances.

Evidence level 4

**'All-fours' technique:** the 'all-fours' position has been described, with an 83% success rate in one case series.<sup>69</sup>

Evidence level 3

The individual circumstances should guide the healthcare professional as to whether to try the 'all-fours' technique before or after attempting internal rotation and delivery of the posterior arm. For a slim mobile woman without epidural anaesthesia and with a single midwifery attendant, the 'all-fours' position is probably more appropriate, and clearly this may be a useful option in a community setting. For a less mobile woman with epidural anaesthesia in place, internal manoeuvres are more appropriate.

Evidence level 4

### 6.3.3 Persistent failure of first- and second-line manoeuvres: what measures should be taken if first- and second-line manoeuvres fail?

**Third-line manoeuvres should be considered very carefully to avoid unnecessary maternal morbidity and mortality, particularly by inexperienced practitioners.**



Evidence level 3

It is difficult to recommend an absolute time limit for the management of shoulder dystocia as there are no conclusive data available, but there appears to be a very low rate of hypoxic ischaemic injury up to five minutes.<sup>51</sup>

Several third-line methods have been described for those cases resistant to all standard measures. These include cleidotomy (surgical division of the clavicle or bending with a finger), symphysiotomy (dividing the anterior fibres of symphyseal ligament) and the Zavanelli manoeuvre. It is rare that these are required.

Vaginal replacement of the head (Zavanelli manoeuvre), and then delivery by caesarean section has been described<sup>70,71</sup> but success rates vary.<sup>72</sup> Intuitively, the Zavanelli manoeuvre may be most appropriate for rare bilateral shoulder dystocia, where both the shoulders impact on the pelvic inlet, anteriorly above the pubic symphysis and posteriorly on the sacral promontory. The maternal safety of this procedure is unknown, however, and this should be borne in mind, knowing that a high proportion of fetuses have irreversible hypoxia-acidosis by this stage, and it may not reduce the risk of BPI.<sup>73</sup>

Evidence level 4

Similarly, symphysiotomy has been suggested as a potentially useful procedure, both in the developing<sup>74,75</sup> and developed world.<sup>76</sup> However, there is a high incidence of serious maternal morbidity and poor neonatal outcome.<sup>77</sup> Serious consideration should be given to these facts, particularly where practitioners are not trained in the technique.

Evidence level 4

Other techniques, including the use of a posterior axillary sling, have been recently reported but there are few data available.<sup>78,79</sup>

#### *6.4 What is the optimal management of the woman and baby after shoulder dystocia?*

##### **Birth attendants should be alert to the possibility of postpartum haemorrhage and severe perineal tears.**



There is significant maternal morbidity associated with shoulder dystocia, particularly postpartum haemorrhage (11%) and third and fourth degree perineal tears (3.8%).<sup>11</sup> Other reported complications include vaginal lacerations,<sup>80</sup> cervical tears, bladder rupture, uterine rupture, symphyseal separation, sacroiliac joint dislocation and lateral femoral cutaneous neuropathy.<sup>81,82</sup>

Evidence level 2+ and Evidence level 3

##### **The baby should be examined for injury by a neonatal clinician.**



BPI is one of the most important complications of shoulder dystocia, complicating 2.3% to 16% of such deliveries.<sup>7,11,13,14</sup>

Other reported fetal injuries associated with shoulder dystocia include fractures of the humerus and clavicle, pneumothoraces and hypoxic brain damage.<sup>15,83,84</sup>

Evidence level 3

##### **An explanation of the delivery should be given to the parents (see section 9).**



#### **7. Risk management**

##### *7.1 Training*

###### **7.1.1 What are the recommendations for training?**

###### **All maternity staff should participate in shoulder dystocia training at least annually. Grade D**



The fifth CESDI report recommended that a 'high level of awareness and training for all birth attendants' should be observed.<sup>50</sup> Annual 'skill drills', including shoulder dystocia, are recommended jointly by both the Royal College of Midwives and the RCOG<sup>85</sup> and are one of the requirements in the Clinical Negligence Scheme for Trusts (CNST) maternity standards.<sup>86</sup>

Evidence level 4

Where training has been associated with improvements in neonatal outcome, all staff received annual training.<sup>14</sup>

Evidence level 3

One study looked at retention of skill for up to one year following training using simulation. If staff had the ability to manage a severe shoulder dystocia immediately following training, the ability to deliver tended to be maintained at one year.<sup>97</sup>

Evidence level 2-

###### **7.1.2 What is the evidence for the effectiveness of shoulder dystocia training?**

Practical shoulder dystocia training has been shown to improve knowledge,<sup>88</sup> confidence<sup>89</sup> and management of simulated shoulder dystocia.<sup>90-93</sup> Training has also been shown to improve the actor patients' perception of their care during simulated shoulder dystocia.<sup>94</sup>

Evidence level 1-

The effect of training on actual perinatal outcomes have been variable: an eight year retrospective review of shoulder dystocia management before and after the introduction of annual shoulder dystocia training for all staff in one UK hospital demonstrated a significant reduction in neonatal injury at birth following shoulder dystocia (9.3% pre-training, 2.3% post-training).<sup>14</sup> There are other reports of improvements after training,<sup>53,95</sup> although in one recent USA study<sup>95</sup> there was increase in the caesarean section rate - from a pre-training rate of 29.90% to a post-training rate of 40.14% - which could account for at least some of the effect.

Evidence level 3

However, training has also been associated with no change in outcome<sup>96</sup> or even deterioration in neonatal outcome;<sup>97</sup> hospitals should therefore monitor the neonatal injury rate after the introduction of training to ensure it is effective.

#### 7.1.3 What measures can be taken to ensure optimal management of shoulder dystocia?

**Manoeuvres should be demonstrated in direct view, as they are complex and difficult to understand by description alone.**



**Higher fidelity training equipment should be used.**



Practical training using mannequins has been associated with improvements in management in simulation<sup>90-93</sup> and in real life.<sup>14</sup>

The largest trial of shoulder dystocia training found that before training only 43% of midwives and doctors could successfully manage a severe shoulder dystocia simulation within five minutes.<sup>91</sup> Three weeks after a 40 minute simulation training session 83% of staff were able to successfully complete the delivery. Training on a high fidelity mannequin was more successful than training with lower fidelity rag doll and pelvis - with a significantly higher successful delivery rate (95% versus 72%), a shorter head-to-body interval and a lower total applied force successful delivery rate.<sup>91</sup>

Moreover, the traction used in simulated shoulder dystocia can be excessive<sup>98,99</sup> but training using models with force monitoring can reduce the traction used in simulated shoulder dystocia.<sup>98,100,101</sup>

Shoulder dystocia training associated with improvements in clinical management and neonatal outcomes was multi-professional, with manoeuvres demonstrated and practiced on a high fidelity mannequin.<sup>14</sup> Teaching used the RCOG algorithm (see appendix 2) rather than staff being taught mnemonics (e.g. HELPERR) or eponyms (e.g. Rubin's and Woods' screw).

#### 7.2 Documentation

**Documentation should be accurate and comprehensive. GPP**



The sixth CESDI annual report highlighted inadequate documentation in obstetrics, with potential medico-legal consequences.<sup>102</sup> Poor documentation of shoulder dystocia management has been highlighted<sup>103,104</sup> and it has been suggested that documentation should be included in shoulder dystocia training.<sup>105</sup> The use of a structured pro forma has been suggested to improve accurate record keeping in the clinical setting<sup>5</sup> and there is some evidence that they are effective.<sup>106</sup>

Evidence level 2+

An example is provided in appendix 3.

It is important to record within the birth record the:

- time of delivery of the head and time of delivery of the body
- anterior shoulder at the time of the dystocia

- manoeuvres performed, their timing and sequence
- maternal perineal and vaginal examination
- estimated blood loss
- staff in attendance and the time they arrived
- general condition of the baby (Apgar score)
- umbilical cord blood acid-base measurements
- neonatal assessment of the baby<sup>104,106</sup>

It is particularly important to document the position of the fetal head at delivery as this facilitates identification of the anterior and posterior shoulder during the delivery.

#### 8. Suggested audit topics

- incident reporting of shoulder dystocia (CNST standard)
- critical analysis of manoeuvres used in the management of shoulder dystocia
- neonatal team called at diagnosis of shoulder dystocia
- documentation of the event (see above)
- performance of cord blood gas analysis
- monitoring neonatal injury (BPI bony fractures) following shoulder dystocia
- staff attendance at annual training
- discussion of events with parents.

#### 9. Support

An information leaflet for parents 'A difficult birth: what is shoulder dystocia?' produced by the RCOG is available online (<http://www.rcog.org.uk/womens-health/clinical-guidance/difficult-birth-what-shoulder-dystocia>).

The Erb's Palsy Group ([www.erbspalsygroup.co.uk](http://www.erbspalsygroup.co.uk)) provides an excellent support network for children and families affected by BPI.

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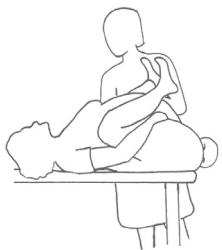
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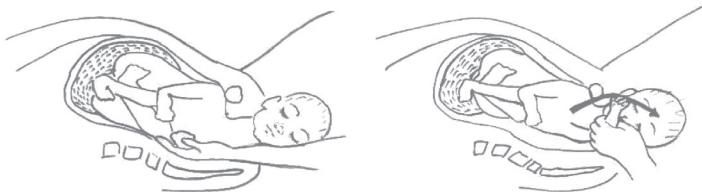
APPENDIX 1



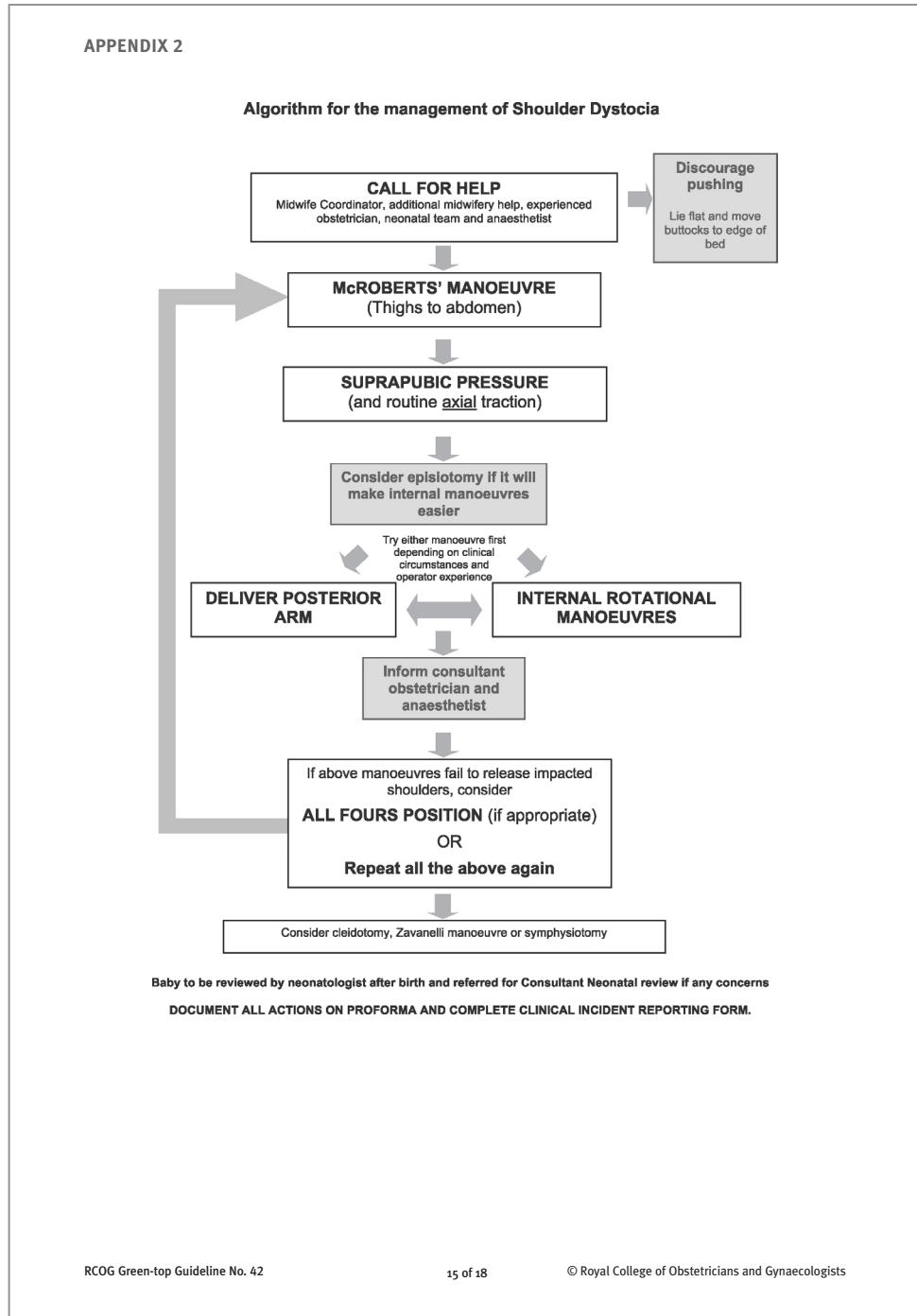
**Figure 1.** The McRoberts' manoeuvre (from the SaFE study)



**Figure 2** Suprapubic pressure (from SaFE study)



**Figure 3** Delivery of the posterior arm (from the SaFE study)



## APPENDIX 3

## SHOULDER DYSTOCIA DOCUMENTATION

Date .....	Mother's Name .....
Time .....	Date of birth .....
Person completing form .....	Hospital Number .....
Signature .....	Consultant .....

Called for help at:		Emergency call via switchboard at:		
Staff present at delivery of head:		Additional staff attending for delivery of shoulders		
Name	Role	Name	Role	Time arrived

Procedures used to assist delivery	By whom	Time	Order	Details	Reason if not performed
McRoberts' position					
Suprapubic pressure				From maternal left / right (circle as appropriate)	
Episiotomy				Enough access / tear present /already performed (circle as appropriate)	
Delivery of posterior arm				Right / left arm (circle as appropriate)	
Internal rotational manoeuvre					
Description of rotation					
Description of traction	Routine axial (as in normal vaginal delivery)	Other -		Reason if not routine axial:	
Other manoeuvres used					

Mode of delivery of head		Spontaneous		Instrumental – vacuum / forceps	
Time of delivery of head		Time of delivery of baby		Head-to-body delivery interval	
Fetal position during dystocia		Head facing maternal left Left fetal shoulder anterior		Head facing maternal right Right fetal shoulder anterior	
Birth weight	kg	Apgar	1 min :	5 mins :	10 mins :
Cord gases		Art pH :	Art BE:	Venous pH :	Venous BE :
Explanation to parents	Yes	By		AIMS form completed	Yes
Neonatologist called? Yes	Neonatologist arrived: .....	Name: .....			
If neonatologist not called or didn't arrive, give reason: .....					
Baby assessment after birth (maybe done by M/W): Any sign of arm weakness? Any sign of potential bony fracture? Baby admitted to Neonatal Intensive Care Unit? Assessment by .....	Yes	Yes	No	If yes to any of these questions for review and follow up by Consultant neonatologist	

Please copy x 2 copies: x1 maternal notes, x 1 attached to AIMS form.

**APPENDIX 4**

Clinical guidelines are 'systematically developed statements which assist clinicians and women in making decisions about appropriate treatment for specific conditions'. Each guideline is systematically developed using a standardised methodology. Exact details of this process can be found in Clinical Governance Advice No.1: *Development of RCOG Green-top Guidelines* (available on the RCOG website at <http://www.rcog.org.uk/guidelines>). These recommendations are not intended to dictate an exclusive course of management or treatment. They must be evaluated with reference to individual patient needs, resources and limitations unique to the institution and variations in local populations. It is hoped that this process of local ownership will help to incorporate these guidelines into routine practice. Attention is drawn to areas of clinical uncertainty where further research might be indicated.

The evidence used in this guideline was graded using the scheme below and the recommendations formulated in a similar fashion with a standardised grading scheme.

<b>Classification of evidence levels</b>	<b>Grades of recommendations</b>
1++ High-quality meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a very low risk of bias	<b>A</b> At least one meta-analysis, systematic review or randomised controlled trial rated as 1++ and directly applicable to the target population; or A systematic review of randomised controlled trials or a body of evidence consisting principally of studies rated as 1+ directly applicable to the target population and demonstrating overall consistency of results
1+ Well-conducted meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a low risk of bias	<b>B</b> A body of evidence including studies rated as 2++ directly applicable to the target population, and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 1++ or 1+
1- Meta-analyses, systematic reviews of randomised controlled trials or randomised controlled trials with a high risk of bias	<b>C</b> A body of evidence including studies rated as 2+ directly applicable to the target population and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 2++
2++ High-quality systematic reviews of case-control or cohort studies or high-quality case-control or cohort studies with a very low risk of confounding, bias or chance and a high probability that the relationship is causal	<b>D</b> Evidence level 3 or 4; or Extrapolated evidence from studies rated as 2+
2+ Well-conducted case-control or cohort studies with a low risk of confounding, bias or chance and a moderate probability that the relationship is causal	
2- Case-control or cohort studies with a high risk of confounding, bias or chance and a significant risk that the relationship is not causal	
3 Non-analytical studies, e.g. case reports, case series	
4 Expert opinion	<b>Good practice point</b> <input checked="" type="checkbox"/> Recommended best practice based on the clinical experience of the guideline development group

This guideline was produced on behalf of the Royal College of Obstetricians and Gynaecologists by:  
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The guidelines committee lead reviewers were: Dr R Ashe FRCOG, County Antrim, Northern Ireland and Mr M Griffiths FRCOG, Luton.

Conflicts of interest; none declared.

The final version is the responsibility of the guidelines committee of the RCOG.

The guidelines review process will commence in 2015 unless evidence requires an earlier review.

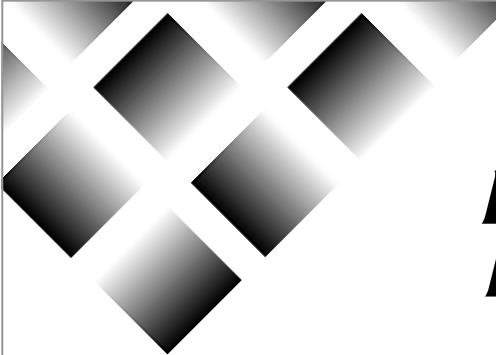
#### DISCLAIMER

The Royal College of Obstetricians and Gynaecologists produces guidelines as an educational aid to good clinical practice. They present recognised methods and techniques of clinical practice, based on published evidence, for consideration by obstetricians and gynaecologists and other relevant health professionals. The ultimate judgement regarding a particular clinical procedure or treatment plan must be made by the doctor or other attendant in the light of clinical data presented by the patient and the diagnostic and treatment options available within the appropriate health services.

This means that RCOG Guidelines are unlike protocols or guidelines issued by employers, as they are not intended to be prescriptive directions defining a single course of management. Departure from the local prescriptive protocols or guidelines should be fully documented in the patient's case notes at the time the relevant decision is taken.

## Appendix B

### The American College of Obstetricians and Gynecologists Practice Bulletin Number 40, *Shoulder Dystocia* ↵



**ACOG**  
**PRACTICE**  
**BULLETIN**

CLINICAL MANAGEMENT GUIDELINES FOR  
OBSTETRICIAN–GYNECOLOGISTS  
NUMBER 40, NOVEMBER 2002

(Replaces Practice Pattern Number 7, October 1997)

## Shoulder Dystocia

This Practice Bulletin was developed by the ACOG Committee on Practice Bulletins—Gynecology with the assistance of Robert J. Sokol, MD, and Sean C. Blackwell, MD. The information is designed to aid practitioners in making decisions about appropriate obstetric and gynecologic care. These guidelines should not be construed as dictating an exclusive course of treatment or procedure. Variations in practice may be warranted based on the needs of the individual patient, resources, and limitations unique to the institution or type of practice.

*Reaffirmed 2013*



**Background**

Shoulder dystocia is most often an unpredictable and unpreventable obstetric emergency. Failure of the shoulders to deliver spontaneously places both the pregnant woman and fetus at risk for injury. Several maneuvers to release impacted shoulders have been developed, but the urgency of this event makes prospective studies impractical for comparing their effectiveness. The purpose of this document is to provide clinicians with information based on published studies regarding management of deliveries at risk for or complicated by shoulder dystocia.

**Maternal Complications**

A study of 236 shoulder dystocia cases reported an 11% rate of postpartum hemorrhage and a 3.8% rate of fourth-degree lacerations (8). These complications were not more common with rotational maneuvers or other fetal manipulations.

lation when compared with the McRoberts maneuver alone (8). It should be noted that the performance of certain “heroic” maneuvers in cases of catastrophic shoulder dystocia, such as the Zavanelli maneuver and symphysiotomy, may be associated with significant maternal morbidity (9, 10).

### **Neonatal Complications**

Brachial plexus injuries and fractures of the clavicle and humerus are associated with shoulder dystocia. The reported incidence of brachial plexus injuries following a delivery complicated by shoulder dystocia varies widely from 4% to 40% (2, 3, 5, 6, 11–18). Fortunately, most cases resolve without permanent disability; that is, fewer than 10% of all cases of shoulder dystocia result in a persistent brachial plexus injury (3, 14–16). Data suggest that a significant proportion (34–47%) of brachial plexus injuries are not associated with shoulder dystocia; in fact, 4% occur after cesarean delivery (11, 19–21).

Some severe cases of shoulder dystocia may result in hypoxic-ischemic encephalopathy and even death (22, 23). A study of outcomes from 6,238 cases of shoulder dystocia found that asphyxia was more common among births complicated by shoulder dystocia regardless of maternal diabetic status (22).

## **Clinical Considerations and Recommendations**

### ► *Can shoulder dystocia be predicted accurately?*

Shoulder dystocia is most often unpredictable and unpreventable. Although fetal macrosomia and maternal diabetes increase the risk of shoulder dystocia (3, 5, 6, 22, 24–28), a substantial proportion of cases occur among women who do not have diabetes and among infants with birth weights less than 4,000 g. In one study, the presence of both diabetes and macrosomia accurately predicted only 55% of cases of shoulder dystocia (5). Additional studies failed to find any combination of risk factors that could accurately predict which pregnancies would be complicated by shoulder dystocia (3, 4, 6, 25, 26). Maternal obesity is associated with macrosomia, and, thus, obese women are at risk for shoulder dystocia. Other antepartum conditions associated with shoulder dystocia include multiparity, postterm gestation, previous history of a macrosomic birth, and a previous history of shoulder dystocia (5, 29). Associated intrapartum factors include labor induction, epidural anesthesia, and operative vaginal delivery (forceps and vacuum-assisted delivery) (3, 4). In each case, risk factors can be identified, but their predictive value is not high enough to be useful in a clinical setting.

### ► *Do labor abnormalities predict shoulder dystocia?*

Three studies have specifically evaluated labor patterns in patients who develop shoulder dystocia (30–32). The largest study, comparing 276 consecutive cases of shoulder dystocia with 600 matched controls, did not identify labor patterns as predictive among any of the cohort, even those with diabetes or macrosomia (30). Another found a significant association between active-phase abnormality and shoulder dystocia, but it included only 36 patients (31). A retrospective analysis of 52 cases of shoulder dystocia reported no difference in protracted dilatation and mean duration of second stage of labor (32). Therefore, data are inadequate to suggest that the labor curve is a useful predictor of shoulder dystocia.

### ► *Does labor induction for suspected fetal macrosomia affect the risk of shoulder dystocia or brachial plexus injury?*

A small, randomized trial of 273 patients with an ultrasound-estimated fetal weight of 4,000–4,500 g comparing labor induction with expectant management reported no significant difference in the rate of shoulder dystocia (3.7% versus 4.3%) or brachial plexus palsy (0% versus 1.4%) (33). Another retrospective study found labor induction with an antenatal diagnosis of macrosomia significantly increased the cesarean delivery rate (36% versus 17%) (34). Labor induction in a woman who does not have diabetes for the sole indication of suspected macrosomia has not been shown to be effective in decreasing the occurrence of shoulder dystocia or decreasing the rate of cesarean delivery (35).

### ► *Is there any benefit to planned cesarean delivery for the prevention of shoulder dystocia in cases of suspected fetal macrosomia?*

A policy of planned cesarean delivery for suspected macrosomic fetuses (>4,000 g) in women who do not have diabetes is not recommended. Ultrasonography is not an accurate predictor of macrosomia (36–38). Furthermore, most macrosomic infants do not experience this complication. Consequently, if all fetuses suspected of being macrosomic underwent cesarean delivery, the cesarean delivery rate would increase disproportionately when compared with the reduction in the rate of shoulder dystocia (6, 24). For example, one study projected a 27% increase in the total cesarean delivery rate (increasing from 15.1% to 19.1%) if cesarean deliveries were performed for all patients with fetuses that weighed 4,000 g or more; unfortunately, the number of shoulder dystocia cases would be reduced by only 42% (6). Another study

reported similar results among fetuses with estimated birth weights of 4,000 g or more; in that study, an additional 76 cesarean deliveries would have prevented only five cases of shoulder dystocia, none of which resulted in permanent injury (39). A study using a decision analysis model estimated an additional 2,345 cesarean deliveries would be required—at a cost of \$4.9 million annually—to prevent one permanent injury resulting from shoulder dystocia if all fetuses suspected of weighing 4,000 g or more underwent cesarean delivery (11). Although the diagnosis of fetal macrosomia is imprecise, prophylactic cesarean delivery may be considered for suspected fetal macrosomia with estimated fetal weights greater than 5,000 g in women without diabetes and greater than 4,500 g in women with diabetes (40).

► ***What should the obstetrician do in cases of shoulder dystocia?***

The performance of the McRoberts maneuver is a reasonable initial maneuver (41). One study described this maneuver as involving hyperflexion and abduction of the hips causing cephalad rotation of the symphysis pubis and flattening of the lumbar lordosis that frees the impacted shoulder (42). Suprapubic pressure may be used at the same time to assist in dislodging the impacted shoulder (1). In contrast, fundal pressure may further worsen impaction of the shoulder and also may result in uterine rupture (12, 17). Controversy exists as to whether episiotomy is necessary, because shoulder dystocia typically is not caused by obstructing soft tissue. Direct fetal manipulation with either rotational maneuvers or delivery of the posterior arm also may be used (43). In these circumstances, performance of a proctoepisiotomy may be helpful to create more room within the posterior vagina.

In cases of severe shoulder dystocia that are not responsive to commonly used maneuvers, more aggressive approaches may be warranted. Cephalic replacement (Zavanelli maneuver) has been described for relieving catastrophic cases (9, 10, 44–46); however, it is associated with a significantly increased risk of fetal morbidity and mortality and maternal morbidity. Intentional fracture of the fetal clavicle may help decrease the bisacromial diameter; however it may be difficult to perform in emergent situations. It is clear that brachial plexus injury can occur regardless of the procedure or procedures used to disimpact the shoulders (3, 4, 47, 48).

► ***How should a woman with a history of delivery complicated by shoulder dystocia be counseled regarding subsequent deliveries?***

A history of shoulder dystocia is associated with a recurrence rate ranging from 1% to 16.7% (3, 26, 49–51).

However, the true incidence may remain unknown because physicians and patients often choose not to attempt a trial of labor when there is a history of a complicated delivery or an injured infant.

Because most subsequent deliveries will not be complicated by shoulder dystocia, the benefit of universal elective cesarean delivery is questionable in patients who have such a history of shoulder dystocia. Other factors that may aid in the decision-making process for mode of delivery include the present estimate of fetal weight compared with the prior pregnancy birth weight, gestational age, the presence of maternal glucose intolerance, and the severity of the prior neonatal injury. A discussion and review of the prior delivery events should be undertaken with the patient, preferably before the intrapartum period. After discussion with the patient, either method of delivery is appropriate.

## **Summary of Recommendations**

*The following recommendations are based on limited or inconsistent scientific evidence (Level B):*

- Shoulder dystocia cannot be predicted or prevented because accurate methods for identifying which fetuses will experience this complication do not exist.
- Elective induction of labor or elective cesarean delivery for all women suspected of carrying a fetus with macrosomia is not appropriate.

*The following recommendations are based primarily on consensus and expert opinion (Level C):*

- In patients with a history of shoulder dystocia, estimated fetal weight, gestational age, maternal glucose intolerance, and the severity of the prior neonatal injury should be evaluated and the risks and benefits of cesarean delivery discussed with the patient.
- Planned cesarean delivery to prevent shoulder dystocia may be considered for suspected fetal macrosomia with estimated fetal weights exceeding 5,000 g in women without diabetes and 4,500 g in women with diabetes.
- There is no evidence that any one maneuver is superior to another in releasing an impacted shoulder or reducing the chance of injury. However, performance of the McRoberts maneuver is a reasonable initial approach.

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The MEDLINE database, the Cochrane Library, and ACOG's own internal resources and documents were used to conduct a literature search to locate relevant articles published between January 1985 and November 2000. The search was restricted to articles published in the English language. Priority was given to articles reporting results of original research, although review articles and commentaries also were consulted. Abstracts of research presented at symposia and scientific conferences were not considered adequate for inclusion in this document. Guidelines published by organizations or institutions such as the National Institutes of Health and the American College of Obstetricians and Gynecologists were reviewed, and additional studies were located by reviewing bibliographies of identified articles. When reliable research was not available, expert opinions from obstetrician-gynecologists were used.

Studies were reviewed and evaluated for quality according to the method outlined by the U.S. Preventive Services Task Force:

- I Evidence obtained from at least one properly designed randomized controlled trial.
- II-1 Evidence obtained from well-designed controlled trials without randomization.
- II-2 Evidence obtained from well-designed cohort or case-control analytic studies, preferably from more than one center or research group.
- II-3 Evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled experiments could also be regarded as this type of evidence.
- III Opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees.

Based on the highest level of evidence found in the data, recommendations are provided and graded according to the following categories:

Level A—Recommendations are based on good and consistent scientific evidence.

Level B—Recommendations are based on limited or inconsistent scientific evidence.

Level C—Recommendations are based primarily on consensus and expert opinion.

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# Appendix C

## The American College of Obstetricians and Gynecologists Patient Safety Checklist Number 6, *Documenting Shoulder Dystocia* ↳47↳55

**The American College of  
Obstetricians and Gynecologists**  
WOMEN'S HEALTH CARE PHYSICIANS



Number 6 • August 2012

### DOCUMENTING SHOULDER DYSTOCIA

Date \_\_\_\_\_ Patient \_\_\_\_\_ Date of birth \_\_\_\_\_ MR # \_\_\_\_\_  
Physician or certified nurse-midwife \_\_\_\_\_ Gravidity/Parity \_\_\_\_\_

Timing:  
Onset of active labor \_\_\_\_\_ Start of second stage \_\_\_\_\_  
Delivery of head \_\_\_\_\_ Time shoulder dystocia recognized and help called \_\_\_\_\_  
Delivery of posterior shoulder \_\_\_\_\_ Delivery of infant \_\_\_\_\_

Antepartum documentation:

Assessment of pelvis

History of prior cesarean delivery: Indication for cesarean delivery: \_\_\_\_\_

History of prior shoulder dystocia       History of gestational diabetes

Largest prior newborn birth weight       Estimated fetal weight \_\_\_\_\_

Cesarean delivery offered if estimated fetal weight greater than 4,500 g (if the patient has diabetes mellitus) or greater than 5,000 g (if patient does not have diabetes mellitus)

Intrapartum documentation:

Mode of delivery of vertex:  
 Spontaneous       Operative delivery: Indication: \_\_\_\_\_  
 Vacuum       Forceps

Anterior shoulder:  
 Right       Left

Traction on vertex:  
 None       Standard

No fundal pressure applied

Maneuvers utilized (1):  
 Hip flexion (McRoberts maneuver)       Suprapubic pressure (stand on the side of the occiput)  
 Delivery of posterior arm       All fours (Gaskin maneuver)  
 Posterior scapula (Woods maneuver)       Anterior scapula (Rubin maneuver)  
 Abdominal delivery       Zavanelli maneuver

Episiotomy:  
 None       Median       Mediolateral       Proctoepisiotomy

Extension of episiotomy:  
 None       Third degree       Fourth degree

Laceration:  
 Third degree       Fourth degree

Cord blood gases sent to the laboratory:  
 Yes: Results: \_\_\_\_\_  
 No

(continued)

*(continued)*

Status of neonate prior to leaving delivery room or operating room:  
 Apgar scores \_\_\_\_\_  
 Evidence of injury \_\_\_\_\_  
 Birth weight (if available) \_\_\_\_\_

Staff present \_\_\_\_\_

Family members present \_\_\_\_\_

Patient and family counseled       Debriefing with appropriate personnel

Postpartum/neonatal documentation:

Delivery discussed with family       Perineal assessment if third or fourth degree laceration

Monitored for postpartum hemorrhage:

Yes: Results: \_\_\_\_\_  
 No

Communication with pediatrics department if there is evidence of injury or asphyxia

Coordination of follow-up care for mother and baby

Monitored for postpartum depression:

Yes: Results: \_\_\_\_\_  
 No

**Procedural Elements for Shoulder Dystocia**

The following steps should be taken when managing shoulder dystocia:

1. Call for help from pediatrics, anesthesia, and neonatal intensive care unit staff, and assign a timekeeper
2. Initiate maneuver (eg, McRoberts maneuver)
3. Re-evaluate course of actions, including using other maneuvers or repeating maneuvers if unsuccessful
4. Consider abdominal delivery
5. Document event—move to documentation checklist

**Reference**

1. Shoulder dystocia. ACOG Practice Bulletin No. 40. American College of Obstetricians and Gynecologists. *Obstet Gynecol* 2002;100:1045–50.

*Standardization of health care processes and reduced variation has been shown to improve outcomes and quality of care. The American College of Obstetricians and Gynecologists has developed a series of Patient Safety Checklists to help facilitate the standardization process. This checklist reflects emerging clinical, scientific and patient safety advances as of the date issued and is subject to change. The information should not be construed as dictating an exclusive course of treatment or procedure to be followed. Although the components of a particular checklist may be adapted to local resources, standardization of checklists within an institution is strongly encouraged.*

**How to Use This Checklist**

The Patient Safety Checklist on Documenting Shoulder Dystocia should be used to guide the documentation process if a patient has experienced shoulder dystocia.

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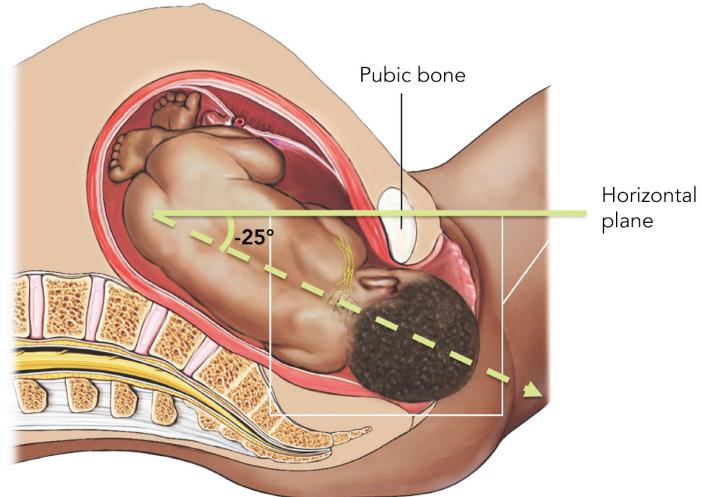
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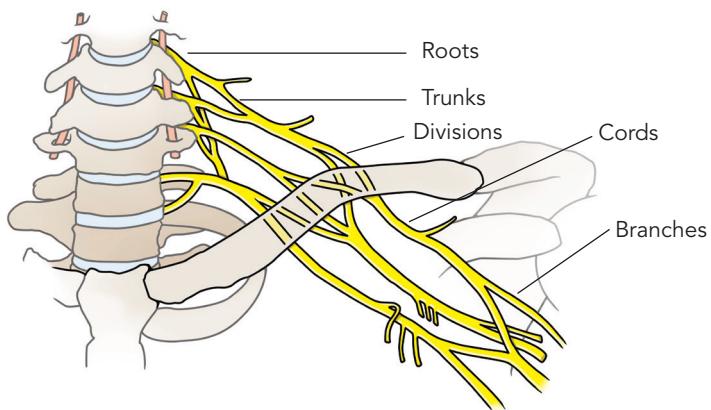
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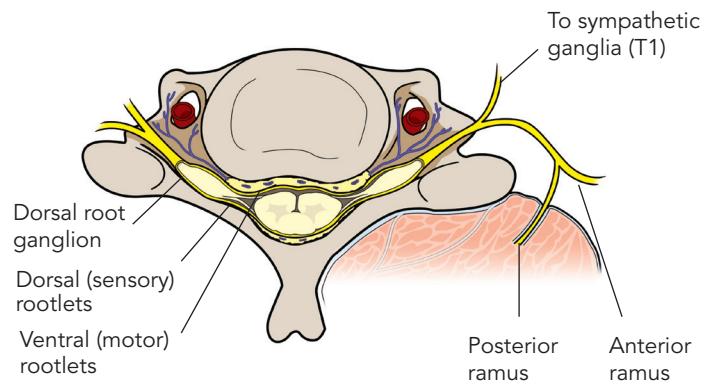
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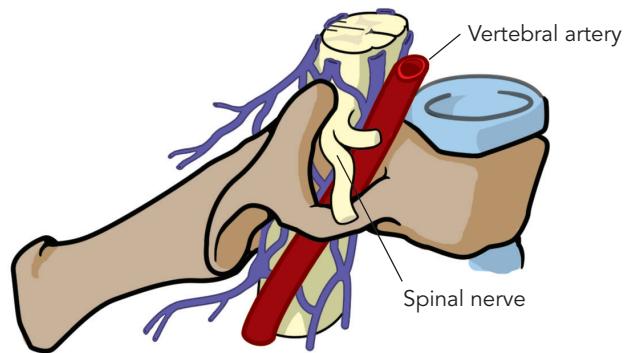
**Figure 3-1.** Traction applied in the plane of the fetal cervicothoracic spine is typically along a vector estimated to be 25–45 degrees below the horizontal plane when the woman in labor is in a lithotomy position. Copyright DoctorStock.com. ↗



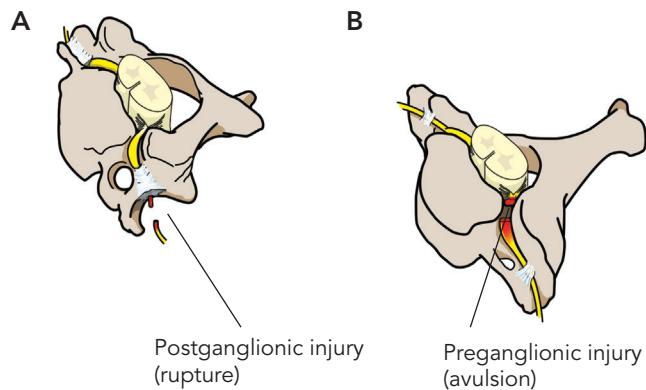
**Figure 5-1.** Schematic diagram of the brachial plexus. ↗



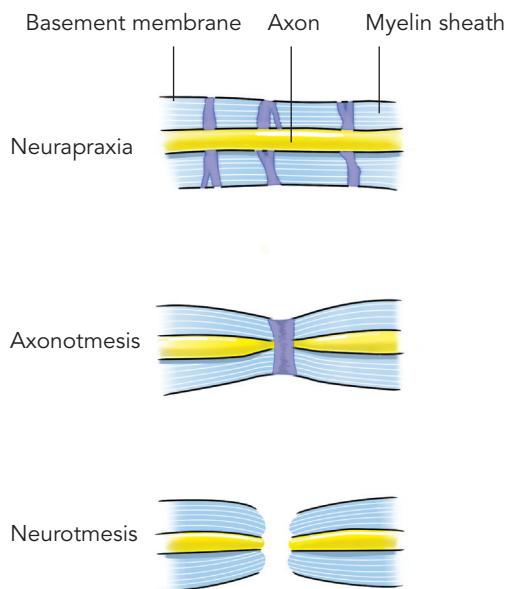
**Figure 5-2.** Axial representation of the spinal column demonstrating the ventral and dorsal rootlets converging into spinal nerve roots and their relationship to the sympathetic ganglia. ↵



**Figure 5-3.** Right lateral view of the cervical vertebra showing relationship of the spinal nerve roots to the vertebral artery (and its surrounding veins) at the level of the neural foramen. ↵



**Figure 5-4.** Diagram of upper (A) and lower (B) cervical vertebrae demonstrating the relationship of the nerve roots as they exit the neural foramina. Because of the inherent differences in the bony foramina between the upper and lower cervical vertebrae, the lower nerve roots more likely sustain preganglionic (avulsion) injury (B). Upper nerve roots more likely sustain postganglionic (rupture) injury (A). ↵



**Figure 5-5.** Seddon classification for nerve injury. ↵

